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## CHOLELITHIASIS (LATENT AND ACTIVE).[1]

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WHETHER it be from the anatomical, physiological or embryological standpoint, the liver should be regarded as a complementary organ to the alimentary canal, the portal system forming the afferent and the biliary tract the efferent pathway—the so-called "hepato-enteric" system of the French.

It has only recently been realized that a morbid condition of the biliary passages constitutes the most frequent type of intraabdominal disease; the great majority of such disorders appear to originate in the gall-bladder and to affect the ducts as a sequel. In fact, it has been estimated that some 40% to 50% of all adults aged over thirty years have disorders of the biliary tract, which may at any time flare up and give rise to active symptoms.

During the past decade an intensive study has been carried out by our medical colleagues into the early stages of such diseases so that the later sequelæ may be prevented.

### ANATOMY.

In a post-graduate lecture only those anatomical details that appear to present a special interest and significance to the surgeon may be dealt with.

[1] A post-graduate lecture, delivered at the Prince Henry Hospital, Sydney, on October 26, 1941.

The gall-bladder, lying in its special fossa on the inferior surface of the right lobe of the liver, is commonly described by British anatomists as being pear-shaped and possessing a fundus, a body and a neck; but, as a rule, there is no neck; a large diverticulum, known as the pelvis or Hartmann's pouch, obscures the origin of the cystic duct, which rises abruptly from the hepatic aspect of the viscus. In other words, the pouch overhangs the origin of the cystic duct, and the exposure of this latter structure may prove difficult when the gall-bladder is acutely distended. Further, "a gall-stone impacted in the cystic duct" will commonly be found on closer examination to be actually a calculus lodged in Hartmann's pouch and coincidentally blocking the opening into the cystic duct—a gall-stone is but rarely impacted in the cystic duct.

The gall-bladder is lined by columnar epithelium, and the mucous membrane is thrown up into a series of folds which subdivide the surface into a series of polygonal areas. Towards the outlet (infundibulum) mucous glands appear, which become more prominent in the pelvis and in the cystic and bile ducts—the gall-bladder can secrete 20 cubic centimetres of mucous fluid a day.

The surgeon should be acquainted with the various anatomical variations in connexion with the gall-bladder, the more common of which have been found to be the following: (*a*) The viscus may be absent, rudimentary, bifid or duplicated. (*b*) It may lie more or less completely embedded in the liver (intrahepatic form). (*c*) Hartmann's pouch may arise from the hepatic instead of the inferior aspect (reversed pelvis). (*d*) The right hepatic duct may empty itself into the gall-bladder. (*e*) Transposition of viscera may be present. In about 10% of cases the gall-bladder is completely invested with peritoneum and possesses a mesentery, so that it swings freely from the inferior surface of the liver.

The cystic duct has a length of one and a half inches and a diameter of one-eighth of an inch, the corresponding measurements in the case of the common bile duct being double the length and two and a half times the diameter of the cystic duct. The lumen of the cystic duct is normally only one-tenth of an inch (2·5 millimetres); but the upper half (*pars valvularis*) is further narrowed by five to twelve corkscrew-like or crescentic folds—the valves of Heister—so that, unless it is dilated, the duct will not permit the passage of a probe. The lower or glabrous part of the cystic duct, together with the hepatic and common bile ducts down to the ampulla, are similar in structure, in that they are simply fibro-elastic tubes with very little muscle.

The common bile duct is tapering in form, gradually diminishing in diameter from one-third of an inch at its origin to one-sixth of an inch at its termination, and may conveniently be divided into three portions. The first, or supraduodenal portion, runs down in the free edge of the lesser or gastro-hepatic omentum and therefore lies anterior to the *foramen epiploicum* (Winslow). The second, or retroduodenal section, passes behind the first (superior) part of the duodenum and then runs downwards in a groove or canal on the posterior surface of the head of the pancreas. The third, or intramural portion, about three-quarters of an inch long, is commonly described as running obliquely through a gridiron-shaped aperture in the duodenal wall, to terminate, in common with the pancreatic duct, in the ampulla of Vater, which sac in turn opens into the duodenal lumen on the summit of the papilla of Vater about four inches (7·0 to 10·0 centimetres) from the pylorus.

Unfortunately, no mention is usually made in the standard text-books of anatomy of those anomalies of the biliary ducts which nevertheless occur with sufficient frequency to be of clinical significance. Such abnormalities readily fall into the following five groups:

1. Variations in the mode of union of the right and left hepatic ducts to form the common hepatic duct.
2. Variations in the mode of union of the cystic and common hepatic ducts. In 17% of persons the cystic duct joins the common hepatic duct at an acute angle in the groove or canal on the posterior surface of the head of the pancreas, after having pursued a more or less parallel course in the right free border of the lesser or gastro-hepatic omentum. In 8% of cases the cystic duct makes a spiral curve either anterior or posterior to the common hepatic duct prior to entering its left border at an acute angle.
3. An accessory hepatic duct which may empty into the right hepatic duct, the common hepatic duct, the cystic duct or even the gall-bladder.
4. Emptying of the right hepatic duct into the cystic duct.
5. Duplication of the cystic duct or the common bile duct.

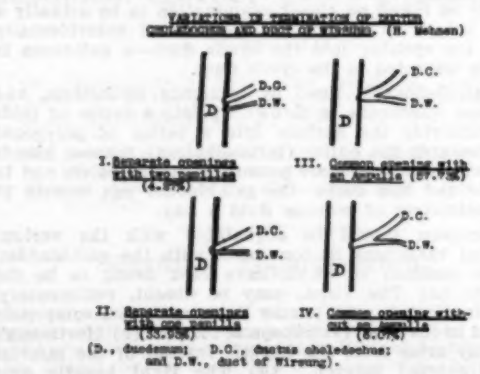


FIGURE I.

Although it is well developed in almost all marsupials, the ampulla of Vater is an atavistic structure, and the tendency in man is for the common bile and pancreatic ducts to open close together and virtually on the mucous surface of the duodenum. In a series of investigations carried out by H. Mehnen, of Berlin, in only 55·73% of cases was an ampulla found of sufficient definiteness to justify the description. The following indicates the relative frequency of the different types of opening: separate openings with two papillæ, 4·27%; separate openings with one papilla, 33·93%; common opening with an ampulla, 55·73%; common opening without an ampulla, 6·07%.

Professor Edward A. Boyden has shown conclusively that the intramural portion of the common bile duct and the associated pancreatic duct are ensheathed in a two-layer *musculus proprius*, which may legitimately be designated as the sphincter of Oddi, after the Italian who first described it in 1886. Although one meets with considerable anatomical variation in the composition of this sphincter, it may be somewhat arbitrarily subdivided into three components.

The *sphincter choledochus* consists of a constant compact sheath of circular fibres encircling the transduodenal portion of the duct as far as its junction with the pancreatic duct. Contraction of this muscle will obstruct the flow of bile, causing the latter to bank up in the gall-bladder, for example, in the intervals between meals. The *sphincter pancreaticus*, present in only one-third of adults, forms an inconstant and less compact annular band surrounding the pancreatic duct just before it joins the bile duct. Around the junction of the two ducts and extending nearly to the orifice of the ampulla is a sheath of delicate circular fibres, the *sphincter ampullæ*; but this is present in only one-sixth of all adults. Less definite sphincteric formations have been described also in connexion with the terminations of the cystic (Keith) and common hepatic ducts (Mirizzi) just prior to their junction.

## PHYSIOLOGY.

It is important to distinguish between the secretion of thin watery bile from the liver, which is a continuous process, and the expulsion of bile into the duodenum, which is intermittent and primarily dependent upon the ingestion of food. When the sphincter of Oddi is contracted the bile ducts fill and bile is diverted into the gall-bladder, where, through the absorption of water and salts, it undergoes great concentration, even down to one-tenth of its volume.

It is these functions of concentration and expulsion that form the physiological basis of cholecystography. If the gall-bladder becomes distended with concentrated bile and can hold no more, the sphincter of Oddi relaxes and the excess of liver bile escapes into the duodenum; but with regular meals the gall-bladder normally can hold all the bile secreted. In other words, the liver produces 500 to 1,200 cubic centimetres in twenty-four hours, and this concentrated ten times will fill the gall-bladder twice or three times in the day; the 20 cubic centimetres of mucus contribute the rest of the gall-bladder contents. Hence in the fasting state the gall-bladder is well filled with concentrated bile, the sphincter is closed and very little bile enters the duodenum, although the liver is constantly secreting it.

The tone of the sphincter of Oddi varies greatly. It is increased with fasting, with distension of the stomach or with the presence of alkali in the stomach, and diminishes with the ingestion of food and with acidity of the gastric contents or with the presence of magnesium sulphate in the duodenum.

Both the muscular wall of the gall-bladder and the sphincter are under the control of nerves derived from the vagal and sympathetic systems. According to Bainbridge and Dale, the vagus is motor to the gall-bladder and inhibitory to the sphincter of Oddi, whilst the sympathetic has the reverse effect. The presence of food in the duodenum (there is only a small psychic flow) causes a reflex contraction of the gall-bladder with reciprocal relaxation of the sphincter, and spurts of concentrated bile issue from the papilla. Of all foods, yolk of egg, fats, including milk and cream, and the vegetable oils are most effective. This activity of the gall-bladder is further augmented by the action of a hormone

(cholecystokinin), which is liberated into the blood stream through the action of hydrochloric acid on the duodenal mucosa. Furthermore, bile salts, absorbed from the intestine, stimulate the liver to secrete larger quantities of bile, which further aids in the digestion of a meal. When digestion is complete these stimuli are no longer present; the sphincter contracts and the gall-bladder begins to refill in preparation for the next meal.

It has been said that the gall-bladder never empties properly when it contains calculi. There is no reason why a calcium bilirubinate stone or a cholesterol "solitaire" should prevent a gall-bladder from emptying, provided the stones lie in the fundus or body. On the other hand, faceted stones, because of the coincident cholecystitis, may well impede the process.

After cholecystectomy the extrahepatic bile ducts dilate, and instead of emerging in intermittent spurts, the bile dribbles away as a continuous trickle from the papilla of Vater. This state is reflected in the results of duodenal intubation. If the sphincter of Oddi is destroyed, no dilatation of the ducts takes place. The intrahepatic ducts are not affected in this dilatation. Similar compensatory changes have been observed in cases of gall-bladder obliteration from disease. Andrews and Hrdina have pointed out that experimental ligature of the cystic duct is almost invariably followed by cholecystitis, whilst ligature of the common bile duct is not. This is an established fact, although difficult of explanation.

## Incidence of Gall-Stones.

Cholelithiasis is a common condition, but its incidence appears to be extremely variable. In Europe every seventh to tenth person has gall-stones, whereas amongst the populations of Japan and the Malay Archipelago the condition is found in only 2% to 4% of the adult population.

Curtis Crump, in a review of 1,000 routine consecutive autopsies conducted at the Pathological Anatomical Institute of the City Hospital in Vienna during the first six months of 1927, states that gall-stones were found in 32·5% of subjects (males, 26·2%; females, 37·8%); some form of abnormal gall-bladder condition in 59·6% (males, 58·4%; females, 60·2%); and a pathological condition of the bile ducts in 41·3% (males, 34·9%; females, 46·5%).

In *The Medical and Scientific Archives of the Adelaide Hospital* (1929), Dr. Ian Hamilton published an analysis of one thousand consecutive post-mortem examinations conducted by the department of pathology of the University of Adelaide between the years 1925 and 1929. The sex incidence was 675 males to 325 females, and gall-stones were discovered in 10·6%, or 106 cases; 64·0% (68 cases) were in males and 36·0% (38 cases) in females. A statistical survey of this series is to be found in THE MEDICAL JOURNAL OF AUSTRALIA of July 16, 1932, and will well repay perusal.

Each year approximately 190 deaths are recorded in the Commonwealth of Australia as being directly due to gall-stones, and of these 39% occur in New South Wales (Table I).

In size gall-stones range from concretions smaller than a millet seed to one the size of a bantam's egg, whilst in number they vary from one to many thousands. Otto reported 7,802 in one patient. As a general rule gall-stones are found much more commonly in women than in men. The close relationship between cholesterol metabolism and ovarian function (compare "the menstrual cholesterol drop") and the hypercholesteræmia following large doses of œstrogenic preparations *et cetera* have been put forward as a possible explanation of this unequal sex ratio.

Gross, reviewing the post-mortem records of the Leeds General Infirmary, found that although gall-stones were twice as common in women as in men, when the proportion of married women to single women (admitted into the hospital) was considered, married women were no more liable to cholelithiasis than single women.

The Mayo Clinic statistics reveal that calculi were limited to the gall-bladder in about 86% of all cases; they were found in the common bile duct in only about 5% and in both gall-bladder and common bile duct in some 7% of cases.

The operative findings would lead one to assess the sex ratio of common-duct stones as nine times in women to once in men.

## Ætiology.

The mode of formation of gall-stones has long been a subject of controversy, and is still by no means fully understood. The fundamental difficulty arises from the fact that the conditions which determine their formation are multiple and complex. In other words, the variation of their appearance, structure and mechanical composition is clear proof of their different modes of origin, and the only satisfactory way to classify them would appear to be to group them according to their chemical composition.

According to the classification of Naunym there are four main types: (i) the common "mixed" or faceted gall-stone, consisting mainly of cholesterol, calcium bilirubinate, and calcium carbonate and having a concentric structure; (ii) the calcium bilirubinate or "mulberry" stone, a soft,

TABLE I.

*Deaths Classified as Due to Biliary Calculi.*

| Year. | Commonwealth of Australia. | | | | | | New South Wales. | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Number of Deaths. | | | Rate per Million of Population. | | | Number of Deaths. | | | Rate per Million of Population. | | |
| | Males. | Females. | Persons. | Males. | Females. | Persons. | Males. | Females. | Persons. | Males. | Females. | Persons. |
| 1930 .. .. | 52 | 131 | 183 | 15·77 | 41·34 | 28·30 | 16 | 47 | 63 | 12·42 | 37·78 | 24·88 |
| 1931 .. .. | 41 | 120 | 161 | 12·34 | 37·44 | 24·67 | 14 | 47 | 61 | 10·78 | 37·37 | 23·87 |
| 1932 .. .. | 42 | 128 | 170 | 12·56 | 39·58 | 25·84 | 16 | 53 | 69 | 12·22 | 41·71 | 26·75 |
| 1933 .. .. | 55 | 156 | 211 | 16·33 | 47·81 | 31·83 | 22 | 63 | 85 | 16·68 | 49·12 | 32·67 |
| 1934 .. .. | 67 | 124 | 191 | 19·77 | 37·69 | 28·60 | 30 | 36 | 66 | 22·57 | 27·81 | 25·16 |
| 1935 .. .. | 67 | 122 | 189 | 19·65 | 36·79 | 28·10 | 32 | 53 | 85 | 23·90 | 40·57 | 32·13 |
| 1936 .. .. | 52 | 122 | 174 | 15·15 | 36·48 | 25·67 | 20 | 45 | 65 | 14·83 | 34·12 | 24·36 |
| 1937 .. .. | 58 | 127 | 185 | 16·77 | 37·63 | 27·07 | 27 | 61 | 88 | 19·84 | 45·75 | 32·66 |
| 1938 .. .. | 61 | 166 | 227 | 17·49 | 48·72 | 32·92 | 20 | 58 | 78 | 14·57 | 43·02 | 28·66 |
| 1939 .. .. | 53 | 135 | 188 | 15·06 | 39·21 | 27·00 | 20 | 45 | 65 | 14·44 | 33·00 | 23·64 |
| Average of five years 1930 to 1934 .. | 51 | 132 | 183 | 15·25 | 40·84 | 27·83 | 20 | 49 | 69 | 15·28 | 38·59 | 25·76 |
| Average of five years 1935 to 1939 .. | 58 | 134 | 192 | 16·75 | 39·68 | 29·20 | 24 | 52 | 76 | 17·63 | 38·97 | 28·19 |

friable, dark brown or green, irregular mass which has been likened to cinders or caviare; (iii) the cholesterol stone ("cholesterol solitaire", "cholesterol monolith" or "*radiarer Cholesterinstein*") of wax-like consistency, oval or rounded in shape, and composed of a conglomerate mass of cholesterol crystals, which are arranged along lines radiating from the centre to its circumference; (iv) biliary pearls, which are small, rounded, multiple calculi composed of radiating crystals of cholesterin and not uncommonly covered by an outer layer of calcium bilirubinate, which gives them a dull brown appearance. Although it is common in animals, the calcium carbonate stone is but rarely found in man.

In this lecture the alleged formation of the common faceted gall-stone and the calcium bilirubinate calculus will be discussed at length.

According to B. Naunym, gall-stone formation was always secondary to local disease of the gall-bladder. This theory assumed that the concretions were formed exclusively in the gall-bladder, that they were the result of local infection and stasis, and finally that the cholesterol of the stone did not originate from the bile, but from the wall of the gall-bladder. Believe it or not, the search for actual proof of this contention did not begin for many years after the theory had been generally accepted. The modern view is that there is some relationship between infection and gall-stones, because of the fact that the gall-bladder wall is infected in approximately 70% of cases of cholelithiasis, the fluid contents are infected in 40%, and the gall-stones themselves are infected in 30% (Gordon Taylor and Whitby—collected statistics). Contrary to the views of A. L. Wilkie and E. C. Rosenow, the common infecting organisms appear to be of intestinal origin, for example, *Bacillus coli*, *Streptococcus fæcalis*, *Bacillus welchii* and *Bacillus typhosus*. Nevertheless, there is little evidence available to determine whether the infection is primary or secondary, or to show the route by which it normally reaches the gall-bladder. Moreover, in quite a number of cases, in spite of the presence of a concretion, the gall-bladder appears to be normal, and even its histological structure may be unchanged.

On the other hand, Chauffard maintains that an increase in the cholesterol content of the blood is responsible for the formation of gall-stones, and holds that sufferers from this malady usually possess a high blood cholesterol level. Other workers have found that an increase is by no means universal, and that the figure is in most cases within normal limits, an increase being constantly present only in those cases of gall-stones associated with jaundice or in which the investigation has been carried out shortly after an attack of gall-stone colic (compare latent jaundice, with blood serum containing between 0·5 and 2·0 milligrammes of bilirubin per 100 cubic centimetres of blood).

It is well known that the common "mixed stone" is composed predominantly of cholesterol, which constitutes from 80% to 98% of the calculus (Peel); that cholesterol may be present in the gall-bladder bile in concentrations between 100 and 900 milligrammes per 100 cubic centimetres (Peel); and that cholesterol may be present in the blood plasma of normal individuals in amounts varying from 80 to 220 milligrammes per 100 cubic centimetres, with an average of 153 milligrammes per 100 cubic centimetres for females and 170 milligrammes for males (Gardner).

Cholesterol is totally insoluble in water, and in bile its state of solution depends upon the presence of bile salts. E. Andrews and his collaborators have shown that if the gall-bladder is inflamed it loses its selective power and the concentrating function becomes abnormal. Its walls then absorb not only water and salts, but also bile acids, which, as has been stated earlier, are mainly responsible for keeping cholesterol in solution.

Further, Andrews, Schoenheimer and Hrdina have shown that the cholesterol content of pooled bile obtained *post mortem* from 30 calculi-containing gall-bladders was 400 milligrammes *per centum*, whereas the bile salt content was no more than 247 milligrammes *per centum*; this yields a bile-salt:cholesterol ratio of 0·6, the normal figure being 20 or more. It remains to be proved, however, whether hypercholesterolæmia will be regularly followed by a coincident increase in the cholesterol content of the bile.

Dostal and Andrews studied the cholesterol content of the bile of a patient with a biliary fistula, and also that of dogs with two-way fistulæ, and found that the cholesterol content of the bile was not influenced by diet, even by a diet containing an excess of pure cholesterol. On such evidence the prescription of a diet poor in cholesterol for patients afflicted with cholelithiasis would appear to be somewhat irrational. Further, since fatty foods are such a potent factor in causing contraction of the gall-bladder (compare cholecystography), there appears to be little reason why such an embargo should be extended to non-calculous cholecystitis.

According to T. Aoyama, the faceted stones are built on a radially arranged protein ground structure, the protein being found mostly in the nucleus and becoming scantier towards the periphery.

With regard to the ætiology of calcium bilirubinate calculi, an enormous number of rod-shaped crystals have been found in the bile *post mortem* in cases of acholuric jaundice, in those of certain diseases of the liver (for example, hæmochromatosis) and in those associated with thin-walled atonic or hypotonic gall-bladders. Furthermore, when bile is allowed to stand either *in vitro* or in the gall-bladder, portion of the bilirubin is precipitated in the form of globular droplets, which are then transformed into similar rod-like crystals. These precipitates are, however, resoluble in fresh liver bile; in this respect they differ from gall-stones, which are quite insoluble in hepatic bile. The presumption is that these irreversible crystals appear as a result of either (i) a secretion of abnormal bilirubin in the bile or (ii) an abnormal process of concentration by the gall-bladder itself.

Few data are available of the time period required for the formation of the common types of human gall-stones; but the structural similarity of different stones from the same patient would suggest that they are formed as a rule at one and the same time—that is, that they belong to the same or to different litters.

Widely varying inflammatory changes may develop in the biliary passages, more especially the gall-bladder, as a result of the presence of a gall-stone. If the calculus is located in the gall-bladder, hyperæmia will be especially evident shortly after an "attack" and generally most obvious in the infundibulum. At other times almost the whole of the mucosa will become involved. If in addition to these inflammatory changes the orifice of the cystic duct becomes occluded, not only the contained bile, but also added products of inflammation collect in the lumen and lead to enlargement of the viscus.

The researches of Professor E. R. Flint and others would indicate that an exacerbation of a chronic calculous cholecystitis produces not only a concomitant hepatitis, but also a coincident inflammatory œdema of the biliary passages. This view is supported by the fact that immediately after an "attack" an increase in the bilirubin content of the blood may frequently be demonstrated, even when there is no perceptible jaundice. Furthermore, even though the cholecystic disease is mild, histological studies of pieces of liver removed at operation from such patients will reveal inflammatory changes in the liver parenchyma as well as in the small bile ducts.

Cholesterosis of the gall-bladder, or the strawberry gall-bladder of Moynihan, is a pathological entity in which the mucosa of the gall-bladder is loaded with lipoid droplets, some in the columnar cells, but most in the histiocytes lying in the stroma of the folds. The majority of gall-bladders so affected show evidence of inflammation, but are sterile on bacteriological examination. Experimentally the strawberry state has been induced in rabbits by combining cholesterol feeding with injection of streptococci into the gall-bladder wall (C. F. W. Illingworth). The mechanism of production would therefore appear to be as follows: infection, absorption of bile salts, precipitation of cholesterol and fatty acids to form a "lipoid mixture", and absorption of the lipoid by the mucosal cells of the gall-bladder.

### SYMPTOMS.

In many cases of cholelithiasis the wall of the gall-bladder and the associated biliary ducts appear, macroscopically, to be quite normal. In a minority of such cases there will have been no obvious clinical symptoms and the calculi are discovered incidentally in the course of a routine examination, at operation for some other condition, or at autopsy. Such patients may be termed "gall-stone carriers"; but they may be readily transformed into "gall-stone sufferers" by the development of superadded inflammatory changes in the biliary passages. In other words, although a small minority of gall-stones may be absolutely silent until a final serious episode, in most instances symptoms do exist which ought to lead to a correct diagnosis in the early stages of the malady.

In a post-graduate lecture the symptoms of cholelithiasis may be best surveyed by, first, a discussion of the typical forms of the disease and, subsequently, a discussion of its atypical manifestations.

### Typical Manifestations.

Calculi in the gall-bladder may give rise to chronic calculous cholecystitis or gall-stone dyspepsia.

#### *Chronic Calculous Cholecystitis.*

The symptoms of chronic calculous cholecystitis are recurrent attacks of aching pain in the right hypochondrium at intervals of days, weeks or possibly years, frequently radiating to the lower angle of the right scapula and commonly associated with irregular vomiting attacks and local tenderness. On deep inspiration there may be a "catch" or pain in the gall-bladder area, and such attacks are not uncommonly mistaken for gastritis. According to the late John B. Murphy, the most characteristic and constant sign of gall-bladder hypersensitivity is the inability of the patient to take a full inspiration when the surgeon's fingers are hooked up deep beneath the right costal arch below the hepatic margin. The diaphragm forces the liver down until the sensitive gall-bladder suddenly reaches the examining fingers, when inspiration suddenly ceases, as though it had been shut off.

#### *Gall-Stone Dyspepsia.*

Gall-stone dyspepsia, or the so-called "inaugural symptoms of gall-stone disease" (Moynihan), gives rise to the following symptoms. There is a sense of fullness, weight and distension or oppression in the epigastrium coming on soon after meals, within half or three-quarters of an hour; it is relieved by belching, dismissed almost instantaneously by vomiting, and elicited with remarkable constancy by certain articles of food, especially those of a greasy nature (a "selective dyspepsia"). As a patient once put it: "Yer feels full up with grub you've never 'ad." In short, three main symptoms may usually be elicited: (i) flatulence, (ii) a feeling of distension after a very little food, and (iii) nausea. In THE MEDICAL JOURNAL OF AUSTRALIA of July 11, 1936, it was pointed out that chronic cholecystitis was frequently associated with achlorhydria, and it is a fair assumption that such symptoms not infrequently originate from a concomitant chronic gastritis.

#### *Calculus Impacted in the Pelvis of the Gall-Bladder.*

A solitary stone, especially a cholesterol solitaire, is apt to gravitate to the pelvis of the gall-bladder and there become permanently or temporarily impacted. If the cystic duct becomes coincidentally obstructed, the gall-bladder is prone to become inflamed, in just the same way as it does after experimental ligation of the cystic duct. A paroxysmal cramp-like pain sets in abruptly, usually at night, and rapidly becomes more severe until in most cases a hypodermic administration of morphine is unavoidable. The pain is most intense below the right costal margin, and from there radiates obliquely upwards towards the inferior angle of the right scapula—that is, it follows the segmental distribution of the eighth thoracic nerve. Murphy's well-known quintet of appendiceal symptoms, occurring in the order of pain, nausea and vomiting, local sensitiveness, elevation of temperature and leucocytosis, is equally applicable to acute inflammatory conditions of this *cul-de-sac* of the biliary tract.

After the abatement of the acute symptoms the protective rigidity gradually subsides, and in a certain number of cases a swelling having the characteristics of a distended gall-bladder makes its appearance. Owing to the coincident hepatitis and associated inflammatory œdema of the intrahepatic ducts, jaundice may occur in a variable percentage of such cases of subacute or acute calculous cholecystitis. As a result of the great discrepancy occurring in the allocation of acute, subacute and chronic cholecystitis as defined respectively by the clinician, the operating surgeon and the pathologist, it must be clearly understood that the above-mentioned differentiation refers solely to the clinical aspect. In other words, it is extremely difficult, often well-nigh impossible, to predict the exact nature and degree of the inflammatory lesion prior to operation; and cases are not infrequently met with in which, in spite of mild clinical symptoms and signs, advanced grades of inflammation of the gall-bladder are revealed at laparotomy.

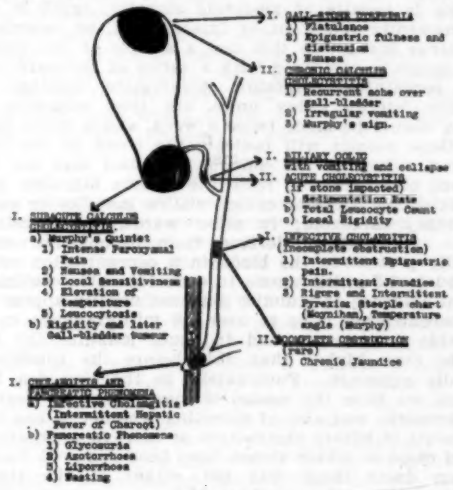


FIGURE II.

A less acute type is the so-called "valve stone", which, although allowing bile to enter the gall-bladder, intermittently prohibits its egress. In such a case cholecystography may give a fair result, whilst duodenal intubation will reveal an intermittent absence of "B" bile.

#### *Cystic Duct Obstruction.*

Owing to the narrow lumen of the cystic duct (one-tenth of an inch) the passage of a calculus along it will initiate a severe type of gall-stone colic.[1] The pain is abrupt in onset, and from the gall-bladder as a centre it radiates in various directions—to the epigastrium and umbilical regions, to the right shoulder and to the inferior angle of the scapula. The agony increases with exacerbations, and lasts (if anodynes are not exhibited) anything from one to ten hours or even longer, when the stone either drops back into the gall-bladder or is passed on into the common bile duct, and the colic abates.

This sudden onset and sudden termination are characteristic of cystic duct obstruction; they never occur in any other form of gall-stone obstruction with anything like the same precision. On the other hand, the stone may become impacted in the duct and cause inflammation, possibly suppuration or even gangrene of the gall-bladder. Biliary colic is to be distinguished from acute cholecystitis by its more acute onset, the greater accompanying collapse and, in the intervals between the exacerbations of colic, by the absence of local abdominal rigidity.

[1] The colic of dyskinesia is characterized by prompt relief following the use of nitroglycerin; calculous colic usually requires the administration of morphine.

*Calculi in the Common Bile Duct.*

Stones in the common bile duct, now believed to be formed in an overwhelming majority of cases in the gall-bladder, must be associated with infection of the ducts from the moment the calculus enters the common bile duct, by reason of the hepatic involvement preceding or following the associated cholecystitis. The symptoms will vary with the degree and type of obstruction produced. They are sometimes trival and inconspicuous; but, sooner or later manifest cholangitis is set up, and pain, jaundice and fever of variable intensity appear. In the majority of cases more than one calculus will be present; the experience of most surgeons is that a solitary stone will be found in about only one case in four. The pain is intermittent, varies much in intensity and is generally restricted to the epigastrium; it is apt to be attended by chilliness and possibly a slight rigor. On deep palpation tenderness is often elicited over the common duct above and to the right of the umbilicus. Mayo Robson's point is about one inch from the umbilicus along a line connecting it with the tip of the ninth right costal cartilage. The jaundice is usually of moderate severity, rapid in onset and frequently remittent or intermittent, as recorded by the icterus index. In this test a sample of the patient's blood serum is compared with a series of standard sealed tubes containing potassium bichromate solution, and arbitrary icterus index units are thus allocated. A reading should be made twice a week, and a curve plotted from these results will indicate the trend of the lesion. However, calculi in the common bile duct may not cause sufficient obstruction to raise the plasma bilirubin to the level at which it either causes visible jaundice or exceeds the kidney threshold; in other words, the jaundice is latent. Bilirubin is derived from hæmoglobin and is normally present in the blood in a concentration varying from 0·1 to 0·3 milligramme in each 100 cubic centimetres. Clinical evidence of jaundice does not usually appear until the concentration rises to over 2·0 milligrammes in each 100 cubic centimetres, and in acute jaundice the figure may be even higher than this before the condition is clinically apparent. Fortunately, in the Van den Bergh reaction we have the means of measuring any degree of bilirubinæmia, and also of detecting whether it has arisen as a result of biliary obstruction or not. In approximately 25% of cases in which stones have been removed from the common ducts there was not, either at the time of operation or in the previous history, any suggestion of clinical jaundice.

It is imperative, therefore, to examine the common bile duct carefully at every operation on the biliary passages, even though there is no history of jaundice.

Rigors with fever of an intermittent or remittent type may occur with or without colic, and may disappear as soon as the infected bile gains entrance into the duodenum. This phenomenon is of serious import, as it indicates infective inflammation of the biliary passages. In other words, a gall-stone located in an infected common duct will give rise to a "hectic" type of temperature like that of a urinary infection, whereas a gall-bladder infection is associated with "septic traumatic fever" recalling that of an abscess.

In rare instances a calculus may completely occlude the common bile duct, giving rise to a rapid onset of intense jaundice, which attains its maximum within a few days. In such cases variations in the intensity of the jaundice, the occurrence of pain or pyrexia and the condition of the gall-bladder are the chief signposts to which to direct our diagnostic acumen. In these late cases the gall-bladder is usually contracted, shrunken and matted down by adhesions, so that no gall-bladder swelling is palpable. This observation was formulated by Courvoisier in the following statement, which is now generally referred to as "Courvoisier's law":

> In cases of chronic jaundice due to blocking of the common duct, a contraction of the gall-bladder signifies that the obstruction is due to stone; a dilatation of the gall-bladder, that the obstruction is due to causes other than stone.

In cases of "silent" jaundice duodenal intubation may afford a valuable diagnostic clue. A dark-coloured "A" bile with an increased concentration of bile salts and the presence of "clumped crystals" microscopically would point to a diagnosis of calculus in the common bile duct. One of the most pronounced and characteristic symptoms of obstruction of the common duct by stone is loss of weight—a loss of two or three stone is not infrequently recorded. Owing to the incomplete digestion and absorption of fats and the loss of bile salts via the urine, the body does not obtain its normal quota of hydrocarbon; hence it is forced to deplete its carbohydrate and amino-acid reserve. Furthermore, chronic pancreatitis is by no means an uncommon event in long-standing obstruction of the common bile duct, whatever the actual site of the obstruction.

*Calculus in the Ampulla of Vater.*

A stone impacted at the ampulla of Vater is a serious menace to the pancreas. The pancreatic secretion may be dammed back into the gland, ascending inflammation of microbic origin of the pancreatic ducts may occur, or bile may find its way into the duct of the viscus, thus activating its enzymes.

Unfortunately, lack of adequate clinical or laboratory tests prohibits the formulation of an exact diagnosis in the great majority of cases of minor degrees of pancreatic deficiency. Temporary glycosuria or an increase in the blood sugar level is not an uncommon accompaniment of gall-stones located in the lower reaches of the common bile duct. Furthermore, our medical colleagues hold that diabetics often give a history indicating previous cholelithiasis.

**Atypical Manifestations.**

*Duodenal Syndrome.*

Chronic cholecystitis, like chronic appendicitis, not infrequently gives rise to reflex pylorospasm, and the symptoms then mimic duodenal ulcer—periodic hunger pain associated with high gastric acidity. No definite radiographic or other signs of duodenal ulcer will, however, be present. Unless such patients are placed on a so-called "post-ulcer régime" subsequent to cholecystectomy, pain and abdominal discomfort are likely to persist.

*Pseudo-Appendiceal Disease.*

It is remarkable how great is the number of patients suffering from either cholelithiasis or duodenal ulcer who present an oblique scar in the right iliac fossa; in fact, it has been facetiously claimed that such an emblem is a necessary adjunct for the diagnosis of the duodenal syndrome and pseudo-appendiceal disease. A small proportion of them have been operated on for acute or subacute appendicitis; but the majority of operations have revealed only "chronic appendicitis", in which the viscus has presented little or no macroscopic evidence of disease. In such cases the "appendiceal" pains may recur after a longer or shorter interval and are at first difficult of explanation, until ultimately it becomes obvious that they are of biliary origin and have been secondarily referred to the ileo-caecal angle.

In rare instances acute appendicitis and acute cholecystitis may coexist; the infecting organisms may be transmitted from the appendix to the liver either through the blood stream or by way of the lymphatics. Here acute appendicitis gives rise to an actual secondary infection in the biliary passages, whereas in the former instance atypical cholelithiasis simulated appendiceal disease.

*"Intestinal Obstruction."*

An interesting condition, which is prone to be overlooked, is that in which the associated cholecystitis has led to the formation of adhesions between the gall-bladder and duodenum with the formation of a cholecysto-duodenal fistula. As the gall-stone escapes to pass down the intestine it is apt to become impacted in the lower end of the ileum and to give rise to acute or subacute intestinal obstruction.

*"Diarrhée Prandiale."*

Reflex disturbances of the colon are occasionally produced by cholelithiasis. Chauffard has pointed out that sudden diarrhœa immediately after meals, sometimes even before they are finished, is not uncommonly associated with gall-stones.

### DIAGNOSIS.

A diagnosis of cholelithiasis is based on four cardinal methods of examination—namely, a careful history, a general physical examination, cholecystography and duodenal intubation. A failure of these methods of approach to reach agreement demands reexamination, not only of the clinical data, but also of the patient. Although in many of the more chronic types of the disease it is quite impossible to do more than make a shrewd guess, one is generally able, in acute cases, to arrive at an accurate diagnosis through ordinary physical signs, more especially if the examination has been repeated at intervals over twenty-four hours or so.

Three methods of approach are at our disposal for the X-ray study of gall-bladder disease: plain films, the opaque meal examination (indirect signs, such as pressure defects, deformity, tenderness *et cetera*) and cholecystography.

Gall-stones impregnated with sufficient calcium may be visualized through a plain X-ray picture.

The introduction of cholecystography by Graham and Cole, in 1924, greatly simplified the recognition of pathological conditions of the gall-bladder; but in spite of more recent improvements this method of examination has as yet attained a proven accuracy of only 80% or 90%. It follows, therefore, that should the results of Graham's test performed after the oral administration of dye prove uncertain when correlated with the clinical findings, the test should be repeated by means of the double dye method of Stewart and Illick, or possibly by the intravenous technique. In short, cholecystography reveals the form and configuration of the gall-bladder and its ability to concentrate and to evacuate. It should, however, be remembered that an absence of cholecystographic shadow may arise either from defective concentrating power or from too little dye-ladened bile entering the gall-bladder. Further, non-opaque ("negative" or "cholesterol") calculi may appear as single or multiple clear-cut, dark, filling defects, usually circular, within the white gall-bladder shadow.

Duodenal intubation, introduced by B. B. Vincent Lyon in 1919, provides bile for analysis and gives information as to the manner in which bile is expelled. To excite the flow of bile from the gall-bladder, 20 cubic centimetres of warm olive oil appear to be the best stimulant. A few minutes after the administration of the oil, bile begins to flow in spurts, at first clear and golden yellow in colour (the "A" or bile-duct bile), then suddenly becoming darker and more viscid (the "B" or gall-bladder bile).

The "B" bile is, of course, not pure gall-bladder bile, but a mixture of gall-bladder bile and liver bile, and normally contains about 300 milligrammes of bilirubin per litre. The "B" fraction flows for about ten minutes, and the bile then again becomes lighter in colour ("C" or hepatic bile). If no concentrated bile is obtained after the repeated use of magnesium sulphate and olive oil, this is presumptive evidence of a non-functioning gall-bladder.

Microscopic and bacteriological examination of the "B" fraction is especially important in those cases in which cholelithiasis is suspected and cholecystography gives inconclusive results. Epithelial débris from the mucus-containing cells lining the infundibulum, as well as pus cells, may be seen in specimens extracted from patients suffering from cholelithiasis and cholecystitis. The finding of "clumped cholesterol crystals" is strong evidence of a cholesterol calculus. The results of the bacteriological tests have to be assessed in conjunction with the gastric and duodenal[1] findings. If these latter specimens are sterile or contain only a few harmless organisms, then any pathogenic strains in the subsequent bile-containing specimens are probably derived from the biliary tract.

Duodenal intubation by the "encapsulated method" of Twiss should therefore reveal the patency of the ducts, the degree of gall-bladder function (as shown by the colour sequence), the probability of local mucosal infection, and the possibility of a calculus.

During the course of an acute exacerbation a weekly or biweekly white blood cell count should be carried out.

Jaundice due to gall-stones has to be distinguished from that due to other causes; one of the most important diagnostic points in favour of a common duct stone is a past history consistent with gall-bladder disease, which may be obtainable in most cases. The diagnosis must necessarily depend upon the clinical history, physical examination, duodenal intubation findings and chemical blood analysis. The jaundice is usually of moderate severity, rapid in onset, and remittent or intermittent as recorded by the icterus index. Clinical icterus is present with an index above 15, whilst a zone of latent jaundice lies between readings of 7·0 and 14·0 units. This test is so simple that if the standard tubes are available it may be performed at the bedside.

During the period of observation, and irrespective of the severity of the jaundice, both bleeding and coagulation times should be determined. An estimation of the blood prothrombin level would appear to be unnecessary unless a definite bleeding tendency is encountered.

### TREATMENT.

From the point of view of treatment, a patient with cholelithiasis has only two alternatives: to keep the stones, together with any pathological disturbances they may be causing, or to have the calculi removed by a surgical operation. Nothing that can be wrought by drugs will have any effect in dissolving the stones.

The prevailing practice of placing all sufferers from cholelithiasis on a "fat-free diet" upon their admission to hospital may be questioned. Such a rationale is apparently based on the erroneous theory of Chauffard, and is aimed at securing a reduction of the cholesterol content of the bile through reducing that of the food. The strongly cholokinetic effect of fat is, however, strikingly demonstrated in cholecystography, and is the foundation of the modern technique of "non-surgical drainage" of the biliary tract, which was introduced by Dr. B. B. Vincent Lyon in 1919. Fats should be cut down only if they lead to pain or dyspepsia, or when jaundice is present.

Theoretically the diet should be high both in Calorie value and in carbohydrate content, and, owing to the deamination function of the liver, it should contain only a minimum quantity of protein. In practice, however, an adaptation of the diet to the individual test-meal findings usually proves much more efficacious. In THE MEDICAL JOURNAL OF AUSTRALIA of November 16, 1940, Professor Henry Priestley pointed out that glucose offered no advantages over starch or cane sugar, and that equivalent amounts of glucose, cane sugar, and boiled or raw starch produced almost identical types of blood sugar curves.

A few years ago it was the fashion to prescribe large doses of hexamine and other allied compounds of ammonia and formaldehyde in combination with sodium bicarbonate and sodium citrate in cases of acute or subacute calculous cholecystitis; but since formaldehyde has never been recovered from the bile obtainable through a duodenal tube (even during a dosage of 300 grains per day), hexamine can no longer be regarded as a biliary antiseptic. Furthermore, it should be remembered that although liver bile is usually alkaline (pH 7·7) and gall-bladder bile slightly acid (pH 6·8), that from an infected tract is commonly alkaline, and so far it has been impossible artificially to change such reaction either by drugs or by diet.

Although a vast array of biliary antiseptics have been advocated from time to time, their therapeutic activity as regards the elimination of infecting organisms from the biliary tract has proved most disappointing. The fact that the condition of many patients has improved clinically

[1] Hanssen and Yuervich have pointed out that the normal duodenum in the fasting state is sterile.

is really no criterion, because a careful follow-up investigation conducted by incubation of the "B" bile reveals that in practically every instance the same organisms persist regardless of treatment. However, recent investigations into the use of sulphanilamide, particularly for patients having persistent post-operative infections, would indicate that this method of treatment may ultimately prove of value.

Whatever "latency" may be invoked by medical treatment in respect of the clinical declarations of gall-stones, there is certainly none attaching to their pathological manifestations. Furthermore, an investigation undertaken in a special clinic (for the study of diseases of the liver and biliary tract) attached to the New York Postgraduate Medical School and Hospital revealed the fact "that medical treatment and the relief of dyskinesia with cessation of symptoms in those patients with stone does not prevent infection due to the presence of stones".

Hence, for the patient with calculi, either in the gall-bladder or in the common bile duct, surgical removal of the stones is necessary to prevent the advent of serious mechanical complications and disastrous infection. In other words, surgical intervention should be strenuously advocated when cholecystography reveals that the concentrating power of the gall-bladder has been lost, when the icterus index is rising, when there is a persistent pathogenic organism in the duodenal bile, and when achlorhydria is present, regardless of any increase or decrease in the subjective symptoms. Unfortunately, gall-stone patients are seldom seen by the surgeon until the disease is well advanced.

In operations upon the biliary passages, it is essential that good access should be obtainable, because such operations, although sometimes easy, may be the most difficult in surgery. A vertical right rectus or paramedian incision is commonly used; but if the bile passages have to be explored, there is no doubt that Kocher's oblique subcostal incision gives better exposure. Several writers on operative surgery describe this last incision as being placed too far laterally, where it does not give anything like such good access. It should commence just below the tip of the xiphoid and run downwards and laterally parallel to, but one and a half ches below, the right costal margin.

In every case the operation begins as an exploration of the whole abdomen, special attention being paid to the stomach, pancreas and appendix; after this the whole biliary apparatus, including the liver, should be carefully examined before any set operative procedure is decided upon. As a routine measure the index finger is introduced into the *foramen epiploicum* and the common bile duct and the head of the pancreas are then thoroughly palpated between the tip of the finger and the thumb.

The general consensus of opinion amongst Australian surgeons is to regard cholecystectomy as the ideal operation for calculi limited to the gall-bladder. The old-fashioned operation of cholecystostomy was too prone to lead to fistula formation or to give rise to a recurrence of symptoms. One of the more recent modifications of cholecystectomy is Professor Max Thorek's operation of electro-surgical obliteration of the gall-bladder, in which the upper wall of the viscus is left attached to its bed in the liver and the lining mucosa is destroyed by bipolar electrocoagulation. In this method it is essential to expose and ligate the cystic duct in order to avoid a biliary fistula. The margins of the retained strip are then approximated with sutures covered by the detached falciform ligament, and the abdomen is closed without drainage.

Whenever possible, especially in routine hospital practice, cultures should be taken of both the contents and the gall-bladder wall during the performance of a cholecystectomy.

When gall-stones are complicated by acute or subacute cholecystitis, the conservative policy of allowing the acute process to subside should be adopted. The vast majority of such lesions respond promptly to therapy on similar lines to that adopted for the Ochsner-Sherren (delayed) treatment of acute appendicitis. Under this régime the patient is placed in the high Fowler's position, nothing but water is given by mouth, adequate fluids are administered subcutaneously, intravenously or by proctoclysis, hot stupes are applied to the region of the gall-bladder and sufficient sedatives are given to ensure comfort, but not to mask the clinical symptoms. Briefly, the method consists of the four "F's": Fowler's position, fluids only, fomentations and four-hourly temperature and pulse recording.

After the acute process has subsided an adequate fluid balance must be maintained and a high carbohydrate intake instituted, so that the general condition of the patient may be so improved as to enable cholecystectomy to be undertaken three or four weeks later. Unfortunately, owing to the intensity of the infection, a small group comprising some 5% to 7% of these lesions will not respond to this conservative treatment, and in such cases an immediate cholecystostomy should be performed as soon as the fluid and sodium chloride balances are restored to normal—say within twenty-four or forty-eight hours. In other words, if the tenderness persists, the temperature remains high, the leucocyte count shows no decrease, or no general improvement is discernible, cholecystostomy with subsequent cholecystectomy at the end of three or four months is the only safe course.

More recently it has been suggested that acute cholecystitis, especially in elderly subjects, often leads to complications which seriously endanger the life of the patient, and on such premises immediate cholecystectomy[1] has been advocated.

The view of the staff of the Prince Henry Hospital is that the mortality rate following early intervention in acute cholecystitis is considerably higher than when a delayed operation is undertaken; further, definite benefit is obtained by the delaying of operative intervention until such time as the general condition of the patient improves and the accompanying pericholecystitis and associated hepatitis[2] have subsided. If such a course is followed, the associated œdema and congestion of the base of the right lung are permitted to subside, and the incidence of post-operative apneumatosis is materially reduced.

Although streptococci, staphylococci and typhoid organisms tend to disappear from the duodenal bile after cholecystectomy, such is not the case with the colon bacillus, which has been found to be particularly resistant to any post-operative treatment. Since this organism has been so commonly found in association with common duct stones, it is believed that such an infection may be one of the factors aiding a recurrence of calculi in this situation.

Since one-tenth of all patients requiring cholecystectomy have calculi located in the extrahepatic biliary passages, provision must always be made for a surgical incursion into these particular passages. In most clinics it is the custom to open and explore all dilated common bile ducts irrespective of whether the patient has a history of obstruction of the duct and regardless of whether a stone can be palpated in it or not. On the other hand, some surgeons restrict exploration of the common duct to cases in which (*a*) a stone can be palpated, (*b*) there is a recent history of jaundice, recurring chills, pain, fever *et cetera*, (*c*) large numbers of calcium bilirubinate crystals or bile sand in association with a persistent colon bacillus infection have been noted in the duodenal bile, or (*d*) jaundice, not otherwise accounted for, is present.

Surgical exploration of the common duct results in a mortality rate over three times as high as that for an uncomplicated cholecystectomy. Whenever possible, a stone in the common duct should be displaced upwards and fixed prior to its being extracted through an incision (Kümmel's supraduodenal choledochotomy) in the middle portion of the supraduodenal segment of the duct. It should be remembered that when a stone is impacted in the lower reaches of the duct, it may be extremely difficult to displace it upwards after the contained bile has been liberated. The high incision in the duct is for the purpose of avoiding the supraduodenal artery of Wilkie

[1] Immediate operation on admission to hospital is by no means synonymous with early operation in the acute attack.

[2] Authorities disagree as to whether this liver damage is a cause, a result or an integral part of the biliary tract condition.

(*rami duodenalis superiores* of Breen). In all such cases the common duct must be proved to be free from calculi; if the duct is dilated and large enough to admit a finger, a digital exploration should be made; otherwise a search should be conducted with a gall-stone scoop or a Desjardins forceps.

A blunt-pointed flexible probe should also be passed down through the papilla into the duodenum. At the same time the probe should also be passed up into the hepatic ducts, for a calculus may have become displaced upwards into one of them.

In an effort to dilate the papilla of Vater many surgeons pass graduated Liston's sounds, commencing with a number 12 and working up to a number 16 or even higher; but the results obtained by such a manœuvre have been shown, by both cholangiograms and post-operative investigations into ductal pressures subsequent to perfusion, to be most transitory. An experimental investigation into the effects of instrumental dilatation of the papilla conducted at the Peter Brent Brigham Hospital showed that no permanent enlargement of the opening resulted and scarring of the papilla took place, whilst earlier still exudation and œdema produced obstruction.

The problem of whether or not the common bile duct should be drained after it has been explored has been the subject of much debate. Some surgeons prefer to close the common duct by interrupted stitches and to insert a drain down to the point of closure. Others[1] insert either a number 12 catheter or a Kehr's T-tube into its lumen and close the remainder of the opening by interrupted stitches.

Notwithstanding these views, direct drainage of the common duct must be employed whenever the contents of the duct are obviously and grossly infected, if the duct contains a quantity of biliary sand or multiple minute calculi, some of which may evade extraction, or on those rare occasions in which it is desired to carry out delayed cholangiography. The T-tube may be removed when the concentration of bile acids in the bile has been restored to normal, when the injection[2] of 10 to 30 cubic centimetres of warm radio-opaque 50% iodized oil (British Pharmacopœia) in sterile olive oil or a 48% solution of hippuran (after aspiration of any residual bile) reveals no obstruction in the vicinity of the ampulla, and when clamping of the tube for forty-eight hours gives rise to no discomfort. If the patient experiences attacks of pain when the T-tube is clamped, say on the eighth to tenth day, a persistent obstruction in or around the duct must be suspected.

With more frequent visualization of the common bile duct subsequent to operation the presence of overlooked stones may often be demonstrated. If the post-operative cholangiogram shows that a common duct stone has been overlooked, Best and Hicken's four-day regimen may be instituted, or an attempt may be made to break up the calculus (Professor B. O. C. Pribram) by instilling one to five cubic centimetres of ether (or ether diluted by alcohol, for example a two to one mixture) into the common duct twice or three times a day. In the latter instance a saturated solution of magnesium sulphate or a crushed tablet of nitroglycerin should be administered orally in order to relax the spasm of the sphincter of Oddi so that the fragments may be expelled into the duodenum.

In rare instances, when at the primary operation it is found impossible to dislodge a calculus from the lower part of the common bile duct, the duodenum should be mobilized and its lumen exposed through a transduodenal approach; the mucous membrane and tissues covering the stone are then incised either at or above the papilla of Vater and the stone is extracted.

It is of great assistance to pass a uterine sound downwards through the supraduodenal choledochostomy incision so as to indicate the position of the duct and at the same time tilt the papilla forwards into the wound.

If the stone be impacted in the lower part of the common bile duct, the duodenum is incised directly over the stone. (Kocher's transduodenal choledochotomy), whereas if the arrest is at the ampulla of Vater it will be necessary only to enlarge the opening (McBurney's duodeno-choledochotomy) by incision of its edges. In the former instance the ductal and posterior duodenal incisions must be approximated by several through-and-through catgut sutures passing through all tissues between the calculus and the duodenal wall. These stitches, therefore, embrace a portion of the bile duct, the intervening compressed and sclerosed pancreatic tissue and the margins of the duodenal incision. The wound in the anterior wall of the duodenum is then closed transversely with the greatest care and securely sealed over with an omental graft.

As the majority of such patients are suffering from cholæmia, infective cholangitis or hepatic insufficiency, the operative risk of surgical removal must necessarily be high, and the mortality rate ranges from 20% to 40% or even more.

Elderly patients who have been severely jaundiced and have the bile passages drained not uncommonly develop a general apathy towards food. In such cases refeeding with bile through an intranasal duodenal tube to the amount of several hundred cubic centimetres daily has been advocated. Since the value of bile for the digestive tract lies in its bile salt content, it would appear far more efficacious to administer by mouth a pharmaceutical preparation of bile salts, such as "Desicol", in the maximum dosage tolerated, rather than to return drainage bile, in which the bile salt concentration may be greatly reduced or even negligible.

As far back as 1933 Professor R. Douglas Wright pointed out that the administration of 4·8 grammes of bile (16 five-grain pills suitably coated with salol) three times a day with meals produced a rise in the blood fat concentration and a pronounced fall in the fæcal fat content, together with great improvement in the general condition of the patient.

### Conclusion.

The surgical viewpoint may be succinctly enunciated as follows.

Gall-stones are never harmless. For long periods they may be latent; but sooner or later, if the host lives long enough, infection and inflammation supervene, with a train of serious complications which may render urgent surgical intervention both difficult and dangerous.

As cholecystectomy in the quiescent period is attended by such a low mortality rate (approximately 3%) and an almost negligible post-operative morbidity, and further, since absence of the gall-bladder causes no evident deterioration in the physical welfare of the patient, surely the obvious corollary is that, unless special contraindications exist, all gall-stones should be removed in the early stages of the malady.

In conclusion, cholecystectomy performed prior to invasion of the biliary ducts practically confers a complete immunity against any recurrence of the stones.

[1] Professor Hans Finsterer advocates a two-centimetre side-to-side or lateral supraduodenal choledochoduodenostomy in such cases.

[2] Oil and bile are not miscible.

## PROBLEMS IN THE THERAPY OF ULCERATIVE GRANULOMA.

By K. VIGORS EARLE, M.D. (London), M.D. (Cambridge), D.T.M. (Sydney),

*Sydney.*

RECENTLY an account of the various diseases to which troops in the Far Eastern theatre of war may be exposed has appeared in the pages of this journal (Poynton, 1941).

Poynton, in his remarks on venereal diseases, mentions ulcerative granuloma (*granuloma venereum*), which, according to him, is the rarest venereal disease. Such rarity is, of course, merely relative, and although practitioners in Australia are for the most part unfamiliar with the condition, they are likely to make its

acquaintance in the future—not necessarily in returned soldiers, but also in civilians. The occurrence of the disease in this latter group may well assume appreciable proportions during the increased commercial and tourist activities in the post-war period.

### Definition.

In the past, loose terminology has resulted in a confusion of ulcerative granuloma (*granuloma venereum*) with *lymphopathia venereum* (also known as *lymphogranuloma inguinale, poradenitis venerea, maladie de Nicholas-Favre, et cetera*). Apart from the suspected venereal origin of the diseases, they have little in common.

According to Rajam (1937), ulcerative granuloma may be defined as "a mildly infective disease of venereal origin, characterized by granulomatous ulceration of the genito-inguinal regions, chronic and progressive in its course without any tendency to spontaneous healing".

### Ætiology.

The disease flourishes among primitive coloured peoples, but can overflow into the civilized coloured races and even into Europeans. In tropical cities it is found amongst the unwashed element—the most degraded coolies, clandestine free-lance prostitutes and inmates of unsupervised brothels

FIGURE I.
Loss of penis by ulcerative granuloma.

of the lowest class. Nevertheless, it can at times mount to the higher strata of society; I recall a lesion—extragenital at that—in a wealthy American *roué* who had acquired it during a tour of the East in search of exotic variations of the sex act.

As the developed lesion is readily recognizable, this disease lends itself to control by the routine inspection of prostitutes, and it appears to be less common in communities practising this form of control than in those where, with pious hypocrisy, the presence of prostitution has no official recognition.

### Clinical Course.

A full description of the course of the disease is beyond the scope of this article. For such, the reader is referred to the excellent descriptions given by Rajam (1937), Manson-Bahr (1935) and Joyeux and Sicé (1937).

If the lesion remains untreated in the male, it proceeds to a loss of the entire penis; the scrotum and adjacent parts of the thigh and abdomen may also receive extensive damage. This type of end-result is seen in Figure I, which shows complete loss of the penis in a young Negro. The man, when seen, had received no specific treatment (he had been given "Salvarsan", since the lesion was thought to be

FIGURE II.
Anterior view. Healing granuloma; new epithelium, pigmented and non-pigmented; œdema of penis, scar of old sinus.

syphilitic). Specific treatment with antimony arrested the disease. In the female the untreated disease leads to gross destruction of the vulva, perineum and rectum, the formation of fistulæ *et cetera*.

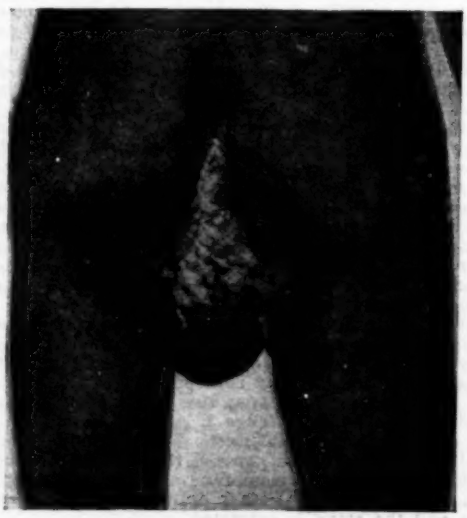

FIGURE III.
Posterior view. Healing granuloma.

Unfortunately, similar disastrous results sometimes occur with inadequate or improper therapy, and even treatment correctly administered may occasionally fail to bring about a cure.

From the therapeutic point of view the lesions can be divided into two groups: (i) non-œdematous and (ii) œdematous.

The first group is represented in Figures II and III, which show the anterior and posterior aspects of a male patient. In this case no œdema occurred in conjunction with the granulomatous tissue, and the lesions, though quite extensive, responded satisfactorily to antimony therapy.

When œdema occurs it may either precede or follow the ulcerative granuloma. In the first instance it is often the result of lymph stasis caused by such conditions as *lymphopathia venereum*, filariasis, chancroid, neoplasm *et cetera*. An example of this type is seen in Figure IV,

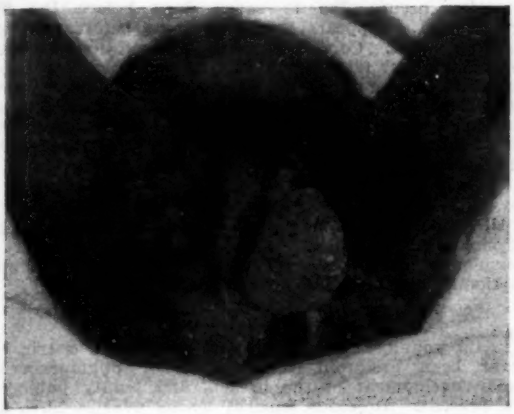

FIGURE IV.
Elephantiasis of vulva; ulcerative granuloma of perineum.

which shows a young Negress with elephantiasis of the vulva caused by *lymphopathia venereum* and further aggravated by pregnancy. Upon this elephantoid tissue is implanted ulcerative granuloma, which not only has attacked the elephantoid tissue, but has also invaded the perineal and perianal areas. In the male, œdematous genitalia, from whatever cause, are also readily attacked; this is shown in Figure V, where commencing destruction of the *glans penis* is illustrated.

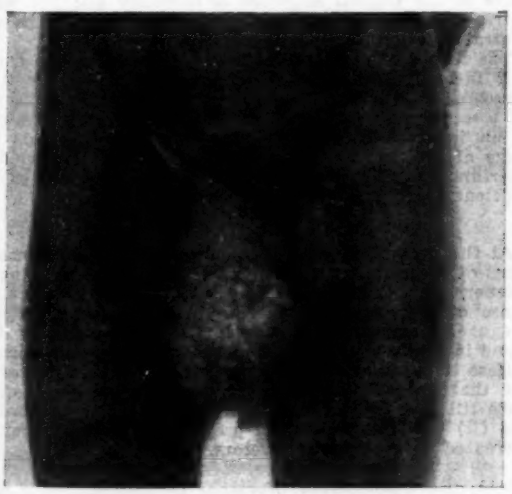

FIGURE V.
Healed granuloma, right groin; erosion of *glans penis* by granuloma.

Œdema may also arise subsequent to ulcerative granuloma as a result of lymph stasis caused by this disease or by inflammation due to secondary infection.

## Treatment.

Only two types of treatment need be considered in this article—antimony derivatives and drugs of the sulphonamide group.

The first antimony compound to be used in this condition was sodium antimonyltartrate (Aragao and Vianna, 1913), and this drug still has considerable vogue, especially in clinics where a low drug bill is of primary importance. Even when correctly given, however, this compound may produce severe joint pain, headache, nausea and vomiting, these manifestations being known as *rheumatisme stibié* (Sézary and Bolgert, 1935). It is scarcely necessary to remark that should any of the injection fluid escape into the paravenous tissues, severe pain, inflammation and perhaps sloughing follow, and should the victim be a clinic patient, a subsequent pronounced dwindling in the total attendance of patients is often noted.

An improvement on sodium antimonyltartrate therapy are the trivalent organic compounds of antimony, of which perhaps "Fouadin" (a complex trivalent antimony salt with a pyrocatechin derivative) is the most extensively used. This compound can be given by intramuscular injections, which are held to be painless; this is not invariably so, since some patients complain of pain at the injection site and also of subsequent arthralgia and nausea.

Under treatment with the newer antimony compounds, especially if these are given in adequate dosage, the majority of granulomata, if not implanted upon œdematous tissue, will clear up. Inadequate dosage may produce an antimony-resistant form of the disease, and occasionally the lesions are resistant to antimony compounds even when these are given in large doses.

When œdema coexists with ulcerative granuloma, especially if such œdema results from a previous infection with *lymphopathia venereum*, treatment yields less satisfactory results. In such types of case antimony compounds, even when given in adequate doses, often fail to effect a cure or even to arrest the progress of the condition. Possibly owing to the fact that the barrier of œdematous tissue allows only inadequate amounts of antimony to act upon the infected tissue, antimony resistance is very apt to develop (Earle, 1938).

Sulphonamide drugs alone do not seem to have any dramatic action upon ulcerative granuloma, although Ross (1939) reports a single case successfully treated with "M & B 693". A combined course of 18 injections of "Fouadin" (given every other day) and "M & B 693" (in a daily dosage of 3·0 grammes for seven to ten days) often produces dramatic results, even in drug-resistant, advanced cases (Earle, 1940). This combined therapy should be given a trial even in apparently hopeless cases.

## Summary.

A short account of ulcerative granuloma is given. Two current types of treatment are discussed and the advantages of combining trivalent antimony derivatives with drugs of the sulphonamide group are pointed out.

## Acknowledgements.

My thanks are due to Mr. J. H. Packer, of Barbados, British West Indies, for the series of photographs illustrating this article.

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## ENDOCRINE CAUSES OF DISORDERS OF MENSTRUATION AND BLEEDING FROM THE NON-GRAVID UTERUS.[1]

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THE field of endrocrinology, more especially that of the sex hormones, has become wider and wider. Its divergence makes it impossible to keep pace with its rapid progress. This is made most difficult for the clinician, as he is endlessly struggling to keep his head above an ever-increasing heap formed of pamphlets thrown at him by the manufacturing chemists, and of small, fragmentary, hurriedly published papers showered upon him by biochemical and biological research workers. If he does manage to emerge, instead of being able to breathe the pure air of understanding, as likely as not he finds himself in a smokescreen of faulty observation and inaccurate deductions produced by his well-meaning clinical brethren. It is little wonder that at times he becomes unreasonable and reckless. Nevertheless all the time new facts are emerging which are gradually correlated to provide a picture of the true endocrine scheme.

Tonight I have assumed that, in the conditions discussed, all causes other than endocrine ones have been systematically excluded, and we are left with none but disturbances of sex hormone equilibrium.

It will be of assistance to make the briefest survey of our knowledge of the endocrine system as it affects the endometrium. The anterior pituitary is looked upon as the controlling body; but it is probable that it is influenced from above perhaps by the hypothalamus. This is particularly shown in amenorrhœa, in which, we are all aware, psychogenic factors often play a not inconsiderable part, operating either through the higher centres or by autonomic control. The anterior lobe of the pituitary produces the following hormonic stimuli: gonadotropic, thyreotropic, adrenotropic, lactogenic, growth, parathyreotropic, pancreatic, diabetogenic and ketogenic. They normally all act in harmony.

The gonadotropic hormones control ovarian periodicity. Prolan A induces ovulation and maturation, and prolan B induces direct luteinization of the follicles even without rupture and ovulation. The ovaries in turn directly control the condition of the uterus during the cycle.

Œstrin is produced in increasing amounts during the pre-ovulatory or proliferative phase as well as during the post-ovulatory or secretory phase. With rupture of the follicle about the thirteenth day there is a sudden and temporary drop in the level of œstrin. Progesterone is produced only after ovulation. The amounts increase till a day or so before the end of the cycle. Œstrin is responsible for the development and growth of the genital tract and for its maintenance at that stage of development. It causes proliferation of the endometrium and inhibits the anterior pituitary. Further, it counteracts progesterone, but is synergistic with it, preparing the uterus for the action of the luteal hormone.

Progesterone changes the proliferative phase of the endometrium to the secretory phase. It counteracts œstrin and may delay ovulation. Progesterone is excreted as pregnandiol. The substance pregnandiol, which is similar in chemical structure to progesterone, is found in the urine only when a *corpus luteum* or a placenta is actively functioning in the body. There is much evidence that it is an excretion product of progesterone metabolism. It is isolated and weighed as a glucuronide complex and expressed in terms of milligrammes of actual pregnandiol. For clinical purposes the total daily excretion is of importance. No pregnandiol is excreted during the proliferative phase of the cycle. During the whole luteal phase of the normal cycle the total varies from three to sixty milligrammes. Pregnandiol first appears in the urine twenty-four to forty-eight hours after ovulation. Maximal excretion occurs five to seven days before the flow. Pregnandiol disappears from the urine before menstrual bleeding starts. Correct pregnandiol excretion estimation depends on four factors: (*a*) the presence of a functioning *corpus luteum* or placenta; (*b*) a normal endometrium to transform progesterone to pregnandiol; (*c*) normal hepatic function to conjugate the pregnandiol; (*d*) normal renal function for its excretion. Failure of any of these factors will lead to an abnormally low reading.

### Diagnosis of Sex Hormone Deficiency.

All endocrine treatment should be based on an accurate physiological diagnosis. It is therefore necessary to consider what tests are at our disposal to demonstrate a particular sex hormone deficiency.

#### *Œstrin Deficiency.*

In the young, in whom adequate amounts of œstrin have never been produced, amenorrhœa or oligomenorrhœa, under-development of the breasts and scanty pubic hair are present. In older women, whose ovaries have once been active, amenorrhœa or oligomenorrhœa, increase of weight and vasomotor disturbances are present.

The epithelium of the vagina is atrophic, with lack of cornification and lack of glycogen; the "secretion" is less acid. The application of Gram's iodine to the epithelium causes a deep brown coloration if the normal amount of glycogen is present in the cells. The degree of brown is proportional to the amount of glycogen in the cells, and it in turn is a measure of the amount of œstrin in the circulation. Examination of smears, instead of revealing large, keratinized, squamous cells with small pyknotic nuclei and few if any leucocytes, reveals fewer and smaller more rounded cells mixed with many leucocytes. The pH value of the vaginal secretion is normally 4·5; but when œstrin deficiency is present the secretion tends to become neutral or even alkaline in reaction.

The length of the uterine cavity is normally two and a half inches. In the presence of œstrin deficiency it is decreased in length. This is not always reliable, however, for a uterus may fail to respond to adequate œstrin owing to an inherent defect. Endometrial biopsy reveals the stage of proliferation of the endometrium. If the endometrium remains atrophic, this is evidence of œstrin deficiency.

#### *Progesterone Deficiency.*

At the present we are unable to recognize any general signs of progesterone deficiency. It may cause bleeding or amenorrhœa. Endometrial biopsy will reveal no secretory phase of the endometrium while the deficiency persists. Pregnandiol glucuronide is absent from the urine, or it is found in the 24-hour output in amounts lower than normal.

Here it is important to remember two things, however: (i) that no pregnandiol is excreted during the normal cycle till twenty-four or forty-eight hours after ovulation, and (ii) that the quantity of pregnandiol excreted may be decreased by extragenital factors.

#### *Gonadotropic Hormone Deficiency.*

The findings of symptoms or signs referable to disorders of the other functions of the anterior pituitary hormones may make one suspect a failure of gonadotropic potency; but this is indefinite and indirect evidence.

Somewhat inaccurate biological quantitative tests exist for the estimation of deficiency in gonadotropic hormone excretion. No definite help can be obtained by estimations

[1] Read at a meeting of the Section of Obstetrics and Gynæcology of the New South Wales Branch of the British Medical Association on June 18, 1941.

of quantities of gonadotropic hormone in the blood owing to their inconsistency.

The mere demonstration of a deficiency of a hormone does not mean that therapy with that particular hormone should be instituted. The time of the finding of the deficiency must first be correlated with the stage of the sexual rhythm. If the deficiency at this stage is abnormal, then there must be a reason to associate it with the patient's symptoms. For example, every woman past the menopause will have a deficiency of œstrin with an atrophic uterus and vagina; but only some will exhibit general symptoms or *kraurosis vulvæ*.

### Disturbances of Sex Hormone Equilibrium.

It will now be as well to consider some of the disturbances of sex hormone equilibrium. The subject will be discussed under the following headings:

1. Menorrhagia and allied uterine bleeding: (*a*) ovular bleeding, (*b*) anovular bleeding, (*c*) bleeding from unproliferated endometrium, (i) at puberty and (ii) after the menopause, (*d*) anovular pseudo-menstruation.
2. Amenorrhœa.
3. The effect on menstruation of disorders of other endocrine glands: (*a*) the thyreoid, (*b*) the suprarenals, (*c*) the pituitary.
4. Granulosa-cell tumours.
5. Hydatidiform mole and chorionepithelioma.
6. Dysmenorrhœa.

#### *Menorrhagia and Allied Uterine Hæmorrhage.*

Menorrhagia and allied uterine hæmorrhage due to endocrine upsets are usually of two types: (*a*) ovular and (*b*) anovular hæmorrhage.

*Ovular Hæmorrhage.*—Ovular hæmorrhage occurs during the middle third of the cycle, generally shortly after ovulation. It is due to a transient lowering of the blood concentration of œstrin between the time of rupture of the follicle and the formation and functioning of the *corpus luteum*. It is similar to an "œstrin withdrawal" hæmorrhage in a castrate under œstrin therapy. The fall in œstrin causes a decrease in endometrial stimulation resulting in a disintegration of the endometrium with hæmorrhage. When the *corpus luteum* commences or increases its production the blood œstrin level rises again, endometrial integration occurs and the hæmorrhage stops. Patients with ovular hæmorrhage usually complain that their menstrual periods are too frequent, usually occurring every two weeks. At times the hæmorrhage is more irregular, as in any one case the fall of blood œstrin level after ovulation is not always of the same degree, and so sometimes the ovular bleeding does not recur in every cycle. The diagnosis is made only after repeated endometrial biopsies are correlated with a time chart of the hæmorrhages or when pregnandiol excretion is similarly correlated. Treatment can be with either œstrin or progesterone. The œstrin administration, properly timed, prevents a fall of the blood œstrin level and maintains endometrial integration, and so no bleeding occurs. Œstrin administration, however, may inhibit the anterior pituitary and delay or inhibit ovulation, so progesterone therapy is preferred. Progesterone, two to five milligrammes, injected every day for two or three days before ovulation is expected, provides stimulation to the endometrium, and it does not disintegrate, but begins to assume the characteristics of the secretory phase.

*Anovular Hæmorrhage.*—Anovular hæmorrhage must not be confused with anovular pseudo-menstruation. In anovular hæmorrhage there is no ovulation, no *corpus luteum* is formed, and a unilateral follicular cyst one or two inches in diameter is present. Hypertrophy of the myometrium and excessive proliferation of the endometrium without any secretory changes are present, otherwise there is cystic glandular hyperplasia. The cells lining the glands are cubical in shape, with central nuclei and contain no glycogen. This is the condition of *metropathia hæmorrhagica*, and is in all probability due to a failure of equilibrium of the pituitary-ovarian mechanism. The follicle fails to rupture, owing to some local condition, or more probably to insufficient stimulation by the anterior pituitary. No *corpus luteum* can be formed, no menstruation can take place. There is a period of amenorrhœa, usually of five to ten weeks' duration, while œstrin is secreted in increasing amounts and the endometrium is increasingly proliferating. Then, possibly because the follicular cyst becomes so tense that there is atrophy of the secretory cells, or because the minute blood vessels in the wall are compressed, œstrin reaches the blood stream in smaller amounts. This causes disintegration of the overgrown proliferative endometrium, and so hæmorrhage. With lowered concentration of œstrin in the blood, the anterior pituitary is less inhibited and so stimulates the ovary to produce more œstrin; this causes integration of the endometrium and the hæmorrhage stops. Thus the hæmorrhage is often intermittent. The diagnosis is made after a consideration of the history, the pelvic findings and endometrial biopsy, together with repeated pregnandiol estimations if required. In this condition also it will be seen that either œstrin or progesterone, suitably administered, will control the hæmorrhage. Progesterone is again preferred. Œstrin injections would temporarily stop the hæmorrhage, but would not permit normal and regular menstruation to be resumed. Furthermore, they would further inhibit the gonadotropic hormone. As no natural *corpus luteum* hormone is already present, progesterone therapy does not require such fine adjustment of dosage, and, being antagonistic to the œstrin, counteracts the depression of the anterior pituitary and allows it to resume its normal function. This it frequently does, and, particularly in the younger subjects, the normal menstrual cycle is resumed. The excessive menstrual flow which occurs soon after the hæmorrhage has been stopped by progesterone is due to the fact that the endometrium has been excessively proliferated throughout the period of the disorder.

*Bleeding from Unproliferated Endometrium.*—At puberty hæmorrhage may occur from an under-developed and infantile uterus with unproliferated endometrium. At or after the menopause hæmorrhage may occur from a small uterus with atrophic endometrium. In these cases the concentration of œstrin in the blood may be too low to produce a proliferative endometrium and too unstable to prevent disintegration from taking place. Now progesterone stimulates only an endometrium which has been prepared sufficiently by œstrin, so it would be useless to give progesterone to control hæmorrhage in such cases, in which there is a low, poorly proliferated endometrium. When hæmorrhage occurs from an infantile uterus with thin endometrium at puberty, œstrin is first given to develop the uterus and increase its size, as well as to prepare the endometrium by proliferation for the later injections of progesterone to produce the secretory phase and later menstruation. It is here hoped that the uterus now being fully developed will respond properly to the cyclical stimulation of the ovaries. At or after the menopause the same treatment may be tried.

*Anovular Pseudo-Menstruation.*—It was observed in monkeys that at times there were regular cyclic discharges of blood which could not be differentiated from normal menstruation (except by examination of the endometrium and of the ovaries), although no ovulation and so no formation of *corpora lutea* had occurred. Such anovular cycles occasionally appear in women. Possibly because of a deficient stimulation by the pituitary the follicle ripens, but no ovulation takes place and an atresic follicle results. The secretion of œstrin is continued by the ovaries and decreases at the usual time, producing cyclical hæmorrhage without ovulation. The hæmorrhage is, of course, from a proliferative and not a secretory endometrium. These cycles may follow one another or may be interspersed amongst normal ovular cycles. In the early cycles following the menarche, and in the cycles during or immediately following lactation, not infrequently ovulation does not occur. Lass, Smelser and Kurzrok, in a recent study of 194 menstrual cycles of 47 women, all initially nursing infants, but with regularly occurring menses, found that 106 were anovulatory and 82 ovulatory, as judged by endometrial biopsies. When the studies were carried on after weaning, the type of result was not affected. It was found that the time of appearance of the first

ovulatory cycle in these women was irregular, and further, that the occurrence of an ovulatory cycle did not predispose them to another ovulatory cycle in the succeeding cycle. Only histological examination of the endometrium and absence of the excretion of pregnandiol can differentiate this anovular pseudo-menstruation from true menstruation.

### *Amenorrhœa.*

The possibility of spontaneous onset of menstruation must always be borne in mind. While treatment need not necessarily be withheld, before a patient is finally classified as amenorrhœic no menstrual periods should have occurred for at least nine months. Indiscriminate treatment without proper prior investigation is to be avoided. All non-endocrine lesions being excluded, the investigation should, when possible, include the taking of a careful history, weighing of the patient, examination of the whole genital tract, with particular reference to the length of the uterine canal, endometrial biopsy, repeated if necessary, X-ray examination of the *sella turcica*, and estimations of the basal metabolic rate, sugar tolerance and hormone excretion.

In more than half the cases the amenorrhœa follows mental trauma or environmental upsets, though on investigation definite hormonal disturbances are found. In treatment the first essential is, as of old, to improve the patient's general health and counteract the psychogenic factor. The return to normal weight is of great importance.

By reading sex hormone pamphlets published by the manufacturing chemists, one is immediately persuaded that œstrogenic therapy is a cure-all and is specific and effective therapy for all cases of amenorrhœa. All it will do is make an atrophic uterus grow, cause the endometrium to proliferate, and produce œstrin withdrawal hæmorrhages. These latter may have the effect of overcoming the psychogenic factor inhibiting ovulation. It is wrong to subject the patient to long and costly treatment if at the conclusion the ovarian function is so poor that the tissues undergo atrophy; it is also harmful, for it is only futher inhibiting the anterior pituitary and depressing what little activity the ovaries may still possess. One must realize that whatever the patient and her relations may think, the only thing that really matters is the return of ovulation.

Œstrin is given if the uterus is undersized or the endometrium scanty, as shown by repeated biopsies. Œstrin may also be exhibited if there is an increased urinary excretion of gonadotropic hormone. This is done on the assumption that the gonadotropic hormone, being produced in an excessive amount, will be inactivated by the œstrin.

The gonadotropic hormone of the serum of pregnant mares is not identical with chorionic anterior pituitary-like hormone. It is more powerful and possesses properties of both the gonadotropic hormones of the pituitary. Serum gonadotropic hormone is capable of stimulating ovarian growth and function in monkeys. It has a follicle-stimulating action, but is also said to produce luteinization in high concentration. Davis and Koff, using it experimentally on women about to undergo laparotomy, claim that under its stimulation follicles can mature, release ova and become *corpora lutea* in from twenty-four to thirty-six hours.

Reliable evidence is lacking that serum gonadotropic hormone is equally effective for patients with hypogonadism. In spite of this, if a normal-sized uterus is present, then serum gonadotropic hormone may be administered, 100 mouse units every three or four days for five injections.

Chorionic hormones used on laboratory animals have mainly a luetinizing effect, and so cannot be expected to influence an unproliferated endometrium such as that frequently found in amenorrhœa; moreover, they have no effect on the ovaries of monkeys. It will be seen, therefore, that they are unlikely to be of help in the treatment of amenorrhœa.

Zondek, Rozin and Vessell used progesterone in the treatment of amenorrhœa. A dosage of 50 milligrammes over ten days produced pseudo-menstruation in 17 out of 19 amenorrhœic women.

### *The Effect on Menstruation of Disorders of Other Endocrine Glands.*

*The Thyreoid.*—Unless cretins are under treatment they do not menstruate. Myxœdema is generally associated with menorrhagia. Joll considers that in hyperthyreoidism menstruation is usually not altered in the early stages, but as the toxicity increases the menses diminish and often disappear. In a few very toxic cases menorrhagia is present.

*The Suprarenals.*—Hyperplasia and neoplasia of the suprarenal glands and Addison's disease all result in amenorrhœa.

*The Pituitary.*—In acromegaly amenorrhœa is present.

### *Granulosa-Cell Tumours.*

Granulosa-cell tumours of the ovary secrete œstrogens in large amounts, so that in young children precocious puberty and menstruation with the corresponding secondary sex manifestations are produced. In adult women periods of amenorrhœa or periodic hæmorrhage from hyperplastic endometrium is produced.

### *Hydatidiform Mole and Chorionepithelioma.*

In hydatidiform mole and chorionepithelioma there is a greatly increased production of anterior-pituitary-like hormone, and pregnancy tests afford a means of diagnosis. After expulsion of a mole the continued excretion of large and increasing amounts of chorionic hormone reveals the presence of chorionepithelioma (often protected from the curette by an intramural position) at a sufficiently early stage for successful surgical removal.

### *Dysmenorrhœa.*

The only pain associated with menstruation of which one of the causes may be an endocrine upset is true intrinsic dysmenorrhœa. All other menstrual pains or menorrhalgia will be uninfluenced by hormone therapy.

True intrinsic dysmenorrhœa occurs in young nulliparous women, and usually dates from two or three years after the menarche. The general health, nutrition and mental outlook are good. The pain is suprapubic, severe and spasmodic, commencing usually about one hour after the onset of the flow and lasting only for the first day. From the endocrine point of view it may be caused by (i) hypoplasia of the uterine muscle, (ii) faulty innervation of the uterine muscle, (iii) a large size of the solid menstrual fragments.

The hypoplasia is demonstrated by the sound, and should be treated by œstrin to increase the growth of the uterus and so improve the function of its muscle.

Faulty innervation of the uterine muscle, according to Kennedy, is due to degenerate changes in the cervical ganglia. He holds that these ganglial changes can be abolished with œstrin. It is possible that the uterine muscle may be too sensitive to stimulation. If this is so, then progesterone may lessen its response.

Large size of solid menstrual fragments, according to Beckwith Whitehouse, is always associated with abnormal and excessive production of decidual cells in the endometrium. He contends that this prevents the normal molecular disintegration and the separation of this tissue, and its presence in the uterus is the cause of the pain. If this excessive decidual reaction is demonstrated, œstrin therapy during the last half of the cycle is indicated.

### Sex Hormone Treatment in General.

It is felt that a few points need stressing.

Correct hormone therapy depends on accurate diagnosis, including a proper knowledge of the nature of the deficiency. Haphazard and unscientific treatment with indiscriminate administration of hormones will not only prove disappointing, but may do considerable harm. Sex hormones, however administered, do not act instantaneously. Even with rapid absorption, the latent period is from twenty-four to forty-eight hours.

The most harmful effect of œstrogenic therapy is depression of the anterior pituitary function, and so of ovarian activity. This particularly applies to the young.

Treatment of young women by œstrogens should never be prolonged more than a few months, and is preferably not given continuously, to minimize this risk.

Male hormones should be avoided. Kerensky, having produced prostate glands in female rats, states that "in the present state of knowledge the results obtained in our experiments and by other workers do not in any circumstances justify the use of the bisexual hormones with pronounced male activity in the treatment of diseases of women".

Testosterone will cause atrophy of the uterine endometrium; but masculinization of the voice, facial hair growth and enlargement of the clitoris have been produced. The danger of a possible pathological perversion of sex feeling exists.

In treatment gonadotropic therapy is not regarded with complete favour. None of the gonadotropic hormones have yet been obtained pure, nor can the degree of impurity of commercial products be estimated. In addition, Frank and Berman, having reviewed the tests available for gonadotropic hormones, conclude that none are more than roughly quantitative.

The reports in the literature on their efficacy are conflicting and mainly subjective. Hamblen sets out the objective observations which should be made when possible, as follows: (i) direct inspection of the ovaries, (ii) direct microscopic examination of the endometrium, (iii) a cytological examination of the vaginal fluid and vaginal epithelial biopsy, (iv) hormonal assays of blood and urine, (v) a search for alterations in the genital system, and (vi) X-ray studies of the skeletal system.

There is no proof that administration of gonadotropic hormones can produce a complete ovarian response. Also, after the exhibition of these gonadotropic hormones, endometrial studies reveal no alteration of the proliferative endometrium in cases of anovular bleeding. It is possible that if an ovarian response has previously been present in a slight degree, it may be increased.

The first essential of all treatment is of course correct diagnosis. In the case of treatment by the sex hormones all other pathological conditions must be carefully and systematically excluded. When that has been done, then the particular primary hormonal dysfunction must be correctly diagnosed. Lastly, the proper active preparation must be administered in the correct dosage, according to the correct time-table, and for the proper length of time.

## COLLAPSE AND RENAL FAILURE.

By J. WALKER TOMB, O.B.E., M.D.,
*Sydney.*

THE cases of renal failure following successful treatment of collapse of the circulation from traumatic shock (now commonly known as "crush injury"), recently reported in *The British Medical Journal*,[1][2][3][4] among patients buried for prolonged periods under débris as a consequence of air raids, bring into prominence the vital importance of an adequate supply of oxygen for normal kidney function.

The term "shock" was originally used by James Latta, of Edinburgh, in 1795, to describe the state of collapse of the circulation following upon severe injury.[5]

McDowall[6] has defined shock as "the state resulting from a fall in arterial blood pressure, which if severe may result in death from oxygen-want". The phenomena of the collapse of the circulation in shock may thus be summarized (after Muir[7]). During shock the capillaries of the skin and abdomen are tightly constricted, while those of the skeletal muscles are dilated and the blood stagnates in them. Blood pressure in consequence falls. The blood vessels of the rest of the body, and particularly the peripheral arteries and veins, are constricted in an effort to maintain blood pressure;[8] abnormal permeability of the walls of the dilated capillaries then develops from want of oxygen[9] and an increased amount of blood plasma passes out of the circulation into the tissues, with the result that the blood becomes concentrated and blood volume is diminished. The amount of blood reaching the right side of the heart thus becomes seriously reduced, and generalized oxygen-want or asphyxiation of the body cells results.

Regarding the increased permeability of the capillaries in shock, Bayliss[10] writes as follows:

> If this increased permeability has not reached too high a value or not been present for too long a time, recovery is possible . . . It is evident, then, that the state is capable of return to normal, if not too serious. The renewed supply of oxygen restores the necessary impermeability to colloids.

Regarding collapse of the circulation as observed in laboratory animals, Bayliss[10] states that if they are treated shortly after the onset recovery takes place, but "if left for two hours or more . . . recovery is impossible" (owing to permanent damage to the capillary endothelial cells).

O'Shaughnessy and Slome[11] make the following statement:

> There is no satisfactory treatment of shock that has persisted for several hours. The subject of severe trauma who does not show some signs of recovery under established modes of treatment within two to three hours of his injury is almost inevitably doomed.

Regarding renal function, Halliburton and McDowall[12] write as follows:

> One fluid, the arterial blood, enters the kidneys; two fluids, the venous blood and the urine, leave it. Both of these are different in composition from arterial blood.
>
> We know that it is not possible to convert any fluid into two others, each of different composition from itself, without an expenditure of energy which must come from somewhere outside the fluids themselves. In the kidney, as in other secreting glands, this energy comes from the cells of the organ and the pressure of the arterial blood.[1] The secretion of urine is therefore the result of work done by the kidney. The quantity of work done may be measured within certain limits and the energy transformed by the kidney may be estimated (in several ways).
>
> Estimations have been made of the amount of oxygen used by the kidney in secreting urines of known concentration. This oxygen may be taken as a measure of the amount of energy used by the organ.
>
> The practical importance of these considerations . . . lies in the fact that the expenditure of energy involves combustion, and combustion demands oxygen. For this reason an efficient supply of oxygen is essential to . . . healthy kidney [function].

With reference to the secretory power of the glandular epithelium of the kidney tubules, Halliburton and McDowall[12] write as follows:

> In frogs the glomeruli can be cut out of action by ligaturing the renal arteries. The kidney is then supplied by the renal-portal vein, a vessel which goes to the tubules only. If urea is then injected under the skin, secretion of urine occurs which, though scanty in amount, is peculiarly rich in urea. Urea therefore in the frog is secreted by the epithelium of the tubules. In order to obtain this result, the kidney must receive sufficient oxygen for the maintenance of the functional activity of its cells. As the arterial supply is cut off by ligature of the renal arteries, this must be accomplished . . . by keeping the frog in an atmosphere of pure oxygen.

Beall *et alii*[1] report that the renal lesion in cases of "crush injury" "consists structurally of severe degenerative changes in the proximal convoluted tubules, and in the more distal parts of the nephron, brown pigmented casts. The matrix of the casts is thought to be composed of desquamated epithelial cells."

A condition similar to the renal failure of "crush injury", without the accompanying hæmaturia due to crushing of soft tissues, but with similar degenerative changes in the convoluted tubules, has long been observed, though hitherto

[1] When the systolic blood pressure falls to 75 millimetres of mercury the secretion of urine ceases (F. R. Winton, *Physiological Reviews*, Volume XVII, 1937, page 408).

unrecognized, in the anuria of cholera persisting after successful restoration of the circulation by infusions of saline solution. Tomb[xi] writes:

The frequent failure of intravenous saline injections to restore and maintain the circulation in cholera, as well as to re-establish the secretion of urine, after collapse has existed for two or more hours, is thus seen to be due to irreparable damage to the capillary endothelium, as well as to the epithelial cells of the kidney tubules, from lack of oxygen.

The histological changes in such cases are thus described by Chatterjee:[xii] "Sometimes—the cells of the tubules show degenerative changes." (In one series of cases the proportion was 30%, no doubt depending inversely on the resistance of the cells of the tubules to tissue asphyxiation.) "These changes consist in fatty degeneration of the [epithelial] cells of the convoluted tubules . . . and also in degenerative swelling of the cells, so that the lumen is obliterated."

Rennie[xiii] has recently drawn attention to two cases of renal lesions from traumatic shock reported in the *Acta Medica Scandinavica*, Volume XCI, 1937, page 279. Both patients sustained fracture of the pelvis and damage to soft tissues. Shock developed in both, yielding to transfusion of citrated blood. Death occurred on the eighth day with a rising blood urea level, hypertension, œdema (in one case) and general signs of uræmia. The urine was very scanty and of low specific gravity, and contained casts and a few erythrocytes; renal function tests gave very poor results. At necropsy no injury to the kidneys or urinary tract was found. Microscopically the chief lesions were emptiness of the glomerular capillaries and degeneration of the epithelium of the tubules, in some of which granular coloured pigment was present.

It would therefore appear that, in so-called "crush injury", the renal failure and associated histological changes in the tubules are caused solely by oxygen-want, as suggested by Sir L. Hill,[xiv] the epithelial cells of the convoluted tubules being even more sensitive to want of oxygen than the endothelium of the blood capillaries itself.

Since the ill effects produced by collapse of the circulation from any cause are chiefly due to tissue asphyxiation of the body cells, the free and uninterrupted exhibition of oxygen in the treatment of collapse and its sequelæ is clearly indicated.

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[viii] W. M. Bayliss: "Intravenous Injections to Replace Blood", Medical Research Council of the Privy Council, Special Report Series Number 25, 1919, page 11.

[ix] L. O'Shaughnessy and D. Slome: "Etiology of Traumatic Shock", *The British Journal of Surgery*, Volume XXII, 1935, page 589.

[x] W. D. Halliburton and R. J. S. McDowall: "Handbook of Physiology and Biochemistry", 1937.

[xi] J. W. Tomb: "Cholera and Uræmia", *The Journal of Tropical Medicine and Hygiene*, July 1, 1941, page 80.

[xii] H. N. Chatterjee: "Histopathology of the Kidney in Cholera", *Transactions of the Royal Society of Tropical Medicine and Hygiene*, Volume XXXIV, Number 4, 1941, page 333.

[xiii] J. B. Rennie: "Crush Injuries", *The British Medical Journal*, Volume I, 1941, page 644.

[xiv] L. Hill: "Crush Injuries", *The British Medical Journal*, Volume I, January, 1941, page 491.


# Reviews.

## SURGICAL NURSING.

DR. H. C. RUTHERFORD DARLING'S well-known text-book on surgical nursing and after-treatment has reached its seventh edition.[1] The first edition was published in 1917, so that the book may well be said to have stood the test of time and to have met a need in the surgical training of nurses. It is three years since the publication of the sixth edition was noted in this journal, and on that occasion the view previously expressed was repeated, that the volume was entirely reliable and could be commended to nurses and others who needed instruction in surgical nursing. The opinion can only be repeated on this occasion, and at the same time we would congratulate the author on his success.

For those who do not know the work, it should be explained that there are two main divisions or sections. The first deals with general surgical nursing and the second with regional surgical nursing. In the first there are useful chapters on bacteriology; immunity and newgrowths (an unusual combination for one chapter); septic infection; ulceration, bed-sores and gangrene; tetanus, gonorrhœa and syphilis; tuberculosis, anthrax, snake bite and burns and scalds (another unusual combination); wounds; surgical technique, ligatures, surgical needles, lotions, powders and drainage materials; the operating theatre; operating in private practice; instruments required for surgical operations; after-treatment; hæmorrhage; shock, syncope, collapse, concussion and cerebral irritation; splints, casings and extension apparatus; fractures, dislocations and sprains; surgical nursing operations; bandaging. In the second section, that on regional surgical nursing, the chapters are devoted to operations on different organs and systems of the body. The chapter on eye operations has been written by Dr. A. James Flynn, and the chapter on ear, nose and throat operations by Dr. R. H. Bettington. Dr. N. D. Royle has contributed the chapter on massage and movements, Dr. Eva A. Shipton has written on the collection of pathological specimens, and Dr. J. G. Edwards has contributed a chapter on preparation for radiological examination and radium therapy. The style of the book is necessarily rather dogmatic, and its value is greatly enhanced by no less than 207 illustrations, which appear to have been carefully chosen. The book is well printed; it is of a convenient size and the binding is all that can be desired.

## THE INTERRELATION OF ABDOMINAL DISEASES.

OBSERVATIONS made over a period of many years on several hundred patients suffering from disease of the alimentary tract and a great many quotations from medical literature are contained in a monograph by E. Forrai on the "Interrelation of Abdominal Diseases".[2] The author points out that chronic cholecystitis is often accompanied by symptoms of disease of other viscera, especially of the stomach, liver and appendix, organs innervated from the same parts of the autonomic system. He claims to show that "gastro-entero-hepato-cholecystitis" is a familial disease; that even isolated manifestations of disorder of stomach, appendix or gall-bladder occurring in members of the same family should arouse suspicion of a constitutional weakness of the whole system, which can often be demonstrated; also that there is a connexion between this disease and familial achlorhydria. He believes that the weak part in the abdominal organic system in these patients is the liver and that when any intercurrent disease occurs which further depresses the function of the liver, such as influenza or thyreotoxicosis, a flaring up of symptoms or else their appearance in a new part of the alimentary canal may be predicted. Allergic disease is also a frequent concomitant, and this is attributed likewise to hepatic disorder in predisposed subjects.

The book lacks cross-headings and it sometimes makes rather heavy reading, as the author tacitly admits by the frequent use of italics for emphasis; the system of references is not uniform, orthodox, complete or satisfactory; but the book is well made and the reader may gain from it a wider and clearer perspective of abdominal disease.

[1] "Surgical Nursing and After-Treatment: A Handbook for Nurses and Others", by H. C. R. Darling, M.D., M.S., F.R.C.S., F.R.F.P.S.; Seventh Edition; 1941. London: J. and A. Churchill Limited. Crown 8vo, pp. 736, with 207 illustrations. Price: 10s. 6d. net.

[2] "Inter-relation of Abdominal Diseases", by E. Forrai, M.D., L.R.C.P., with a foreword by Sir Arthur Hurst, M.A., D.M., F.R.C.P.; 1941. London: William Heinemann (Medical Books) Limited. Demy 8vo, pp. 183, with diagrams. Price: 12s. 6d. net.

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## BERKELEY MOYNIHAN.

IN the George Adlington Syme Oration of 1935 delivered before the Royal Australasian College of Surgeons on the subject of the master surgeon, Professor R. Wood Jones painted a vivid word picture of this impressive personage. He depicted the master surgeons as men cast in a large mould: fine handsome men who lived their lives on a grand scale and who accomplished the fullness of their lives by reason of a boundless energy. He said that there was much truth in the old Italian proverb that "the good surgeon must have the eye of an eagle, the heart of a lion and the hand of a woman". He added that there was running through the lives of these men a thread of feeling that manifested itself at times in formal religion and at times in a deep reverence and a sincere, if not always orthodox, religious sentiment. There was nothing small about the master surgeons; their mental equipment was more than generous and they were indeed men of "great knowledge, great judgement and great humanity". Though we may not approve of the fierce light shed upon them, though we may smile when they are vain, though we may be moved to envy them their eminence, we must acknowledge the enormous debt that surgery and with it all humanity owe to the qualities of heart and hand that are peculiarly theirs. If we say, as we can with truth, that the day of the master surgeon has not passed, thoughts will probably turn at once to the late Berkeley Moynihan, who in his person, his life and his work showed forth many of the qualities and achievements of those extolled by Wood Jones.

The story of Moynihan's life has been told by Dr. Donald Batemen,[1] who was a friend of Moynihan from his Oxford days onwards. It is a very human tale, and reveals Moynihan as a picturesque man with a commanding personality, zealous of his reputation, proud of his achievements, vain perhaps, though not without reason, and last but by no means least, making important contributions to surgical knowledge and arousing British surgeons from a selfish sleepy state to corporate fellowship and united effort. Attention is drawn to this book for two reasons: first of all, to introduce readers to an attractive and well-written biography, and, secondly, to use Moynihan's contributions to surgery as a moral to adorn the tale. Many men and women in medicine who earn as good a living as they can and pose as worthy members of a learned profession, but make no attempt either to increase knowledge or to further the corporate life of their calling, may pause to learn how one man whose life was crowded with incident and crowned with one success after another, was unwilling to seek nothing but his own comfort and well-being. Moynihan made money—conditions of practice made it inevitable that a man of his eminence should—but he could have made more and he might have rested content with wealth, adulation and renown. That he did not is to his lasting credit; he did in fact give almost too much time to affairs outside his practice, but that is to anticipate the story.

Berkeley Moynihan was the son of Andrew Moynihan and of Ellen Anne Parkin, whose father was a cabinet-maker. Andrew Moynihan followed the family tradition by joining the army. He could not afford to seek a commission and so he enlisted as a private in the ninety-sixth regiment. He served in the Crimean War and was the first to receive the newly instituted Victoria Cross. He became Ensign Moynihan and soon received promotion, first to the rank of lieutenant and then to that of captain. It is significant that Andrew Moynihan, in command of "the worst company" of his regiment at Malta, "the blackguards of Number 5", took a personal interest in the welfare of his men, teaching them to read and write, to become efficient and to gain a self-respect that up to the time of his advent had been foreign to them. *Hart's Quarterly Army List* wrote of Andrew Moynihan: "To perfect the training and promote the welfare of the men under his command was an object which absorbed every other interest and to which he devoted all his time, all his thoughts. . . . There is . . . no Company in the British Army more perfect in all its arrangements. . . ." He displayed all the qualities that were later to make famous his son Berkeley George Andrew, who was born at Malta on October 2, 1865. When he died in May, 1867, Mrs. Moynihan with her two little girls and Berkeley return to England. She went to live at Leeds with her sister, whose husband was a police inspector, and they kept house together. Thus began Berkeley Moynihan's association with this provincial centre to which he was to remain true throughout his long and fruitful life and on which he was to shed lustre. At six years of age he first went to school, a "preparatory school for young gentlemen" presided over by three maiden ladies. Later, through the good offices of the Duke of Cambridge, he secured nomination to Christ's Hospital; this was in 1875. He remained here for five years and did not distinguish himself in any way; he left it "unsung, unmissed and fast forgotten" to attend the Royal Naval School at New Cross. Here he did well at his studies and at athletics, equalling

[1] "Berkeley Moynihan, Surgeon", by Donald Bateman; 1941. London: MacMillan and Company, Limited; Sydney: Angus and Robertson, Limited. Medium 8vo, pp. 378, with illustrations. Price: 16s. net.

the amateur record in the one hundred yards swimming race; he also captained the football team. Berkeley intended to enter the army—he had always wanted to be a soldier—but his mother hated the idea. She had set her mind on his being a doctor, but apparently nothing could turn him from his course. It was only when he chanced to overhear his mother pouring out her grief to a friend that he changed his mind, and later he told her that he was going to save life and not take it. Bateman, in justifying Berkeley's sudden change of mind, states that greater doctors than he have been impelled by lesser inspiration. "In medicine the spirit grows by what it feeds upon. Increased awareness of the range and horror of disease awakes the student's charity. Out of this springs true devotion." That this was true in young Berkeley's case is abundantly clear from Bateman's narrative. By the generosity of two uncles, Berkeley was able to start his medical course at Yorkshire College and the Leeds General Infirmary. Students at Yorkshire College (it ultimately became the University of Leeds) could sit for the examinations of the University of London, and this Berkeley did. He graduated in 1887 and had passed the primary fellowship examination of the Royal College of Surgeons of England during his undergraduate course. Before long his feet were set on the road to consulting practice as a surgeon—the road to success.

Moynihan's contributions to surgery may be grouped under two headings: those that he made to surgical knowledge and practice, and those that he made to the union, the communion, of British surgeons and to British surgery as a vital corporate entity. Before these are mentioned some reference must be made to Isabel his wife, who gave him companionship, comfort and happiness. She was to him a tower of strength, but we shall allow Bateman to tell us about her. Moynihan needed "someone believing in him to the last, someone to soothe the turbulences of a nature Irish, and of flaming hair—that one he sought, and, at the issue, chose well. . . . Isabel gave to Berkeley a faithful, loving loyalty, and, what he lacked himself, a resilience of spirit that lifted him over the rough and broken ground of all his early years in practice, and, in the more distant years, steadied a mind oft tending to o'ertop itself. . . . The surface of life showed how much Berkeley gave to Isabel—wealth, titles, and success. But let no one suppose the bargain was one-sided. With these gifts came for her burdens, not to be seen by all. And, in the depths, the seeing eye may find revealed those things she gave to him: things not to be held and measured, but which made it possible for Berkeley to obtain much that otherwise might have lain beyond his grasp. She gave him happiness." And so we are ready to look at Moynihan's contributions to surgical knowledge. He did "by an effort set aside a portion of each day for the pursuit of surgery as an art and science, and not as trade alone". Research does not, Bateman points out, necessitate monastic withdrawal into a laboratory. It is in medicine rather an attitude of mind than a circumscribed employment. It does not call for genius; intelligence and application will suffice. Genius is usually responsible for the great advances, but "the researches of intelligence provide the humble, though essential, communications without which the attack could not be made". Moynihan had not the genius of originality; he achieved what he did by scrupulous attention to detail, unflagging interest, and a mind of perfect tidiness. Moynihan's work was chiefly concerned with abdominal conditions, in witness of which we have his books on the pancreas, on the stomach and on gall-stones. If he had done nothing else than give to surgeons his work "Abdominal Operations", his name would surely live. Bateman probably does not overstate the case when he writes that Moynihan blazed the trail along which all abdominal surgeons now march. And we can agree that: "In surgery, though Berkeley stands lower than Lister, the great man's hand yet rests upon his shoulder." Further reference might be made to Moynihan's insistence on the study of what he called the "pathology of the living", to his use of rubber gloves in the face of a supercilious conservatism, but the temptation must be resisted. Two achievements for British surgery must be mentioned: the formation of the Associations of Surgeons and the establishment of *The British Journal of Surgery*. The former was particularly valuable in that it made surgeons throughout England know one another, engage in discussion and study one another's work; it also broke down the fetish that London was the only real home of English surgery. *The British Journal of Surgery* was Moynihan's child and he had as chief assistant at its birth Hey Groves, of Bristol, who is still associated with it. Surgeons were strangely apathetic when the publication of a new journal was mooted. MacEwen, of Glasgow, for example, wrote to say that there was little British surgery of any value and that few men were capable of writing anything worth while. In the first issue there appeared an article on MacEwen's surgical clinic. MacEwen then wrote: "If this is the kind of journal you had in mind I feel sure it will succeed." It did, but not because of special articles about special men and special clinics. Much more remains to be told that cannot be told in this place—of Moynihan's war service, of his meetings with American surgeons, of his lectures in America, of the honours showered upon him, of his vanities and foibles, and of other things too numerous to be mentioned. Readers must seek these in the book itself. Only the closing scene must be touched upon, for it shows that too great a price may be paid for fame, honours and success. Isabel Moynihan, "whose frail body was . . . drawing on its last reserves, was overtaken by sudden collapse" and died on August 31, 1936. Moynihan was sorely stricken, not only by the thought that he must continue in the world alone, but by the memory of all the hours that Isabel had spent alone while he was at his work. This was "a bitter agony to him". "It was a mortal one. The terrible desolation of his mind quickly called upon, and fast destroyed, the last of his physical reserve." Six days after his wife's death he also died.

Berkeley Moynihan must be numbered among the master surgeons. He had a master mind. Though few medical practitioners may be so blest, they can all try to cultivate the methods that are the mark of the master—deliberate search, attention to detail and constant interest. An "infinite capacity for taking pains" comes naturally to a few; there are surely among the others some who may acquire it by diligent effort.

# Current Comment.

## THE CRUSH SYNDROME.

EACH war brings new disease for old. Now, instead of trench maladies, we are faced with the results of blast, direct and indirect, from high explosive aerial bombs. Blast injury to the lung has been well described by Rose in the pages of this journal. Another injury, now called the "crush syndrome", may not yet be so familar to its readers. The title has no relationship to the feeling of a claustrophobe entrapped in an Anzac Day crowd, but represents certain results which follow prolonged pressure on the whole or part of the body by fallen masonry or earth. Such things have occurred before, in mines and dugouts and among tradesmen engaged in house demolishing, but the frequency of such accidents in London has brought certain undesirable, if interesting, consequences under consideration.

D. H. Patey and J. D. Robertson[1] describe the following as a common course for the syndrome. The patient has a history of being buried for several hours under the debris of a bombed house, with compression of a limb or limbs. On his admission to hospital his condition may be good, with normal pulse and blood pressure, but a few hours later shock sets in. Locally œdema appears over and around the compressed area and spreads along the limb or limbs. Movement of the affected limbs may be restricted or absent and various sensory disturbances, such as anæsthesia, hyperæsthesia or paræsthesia may develop. Later oliguria and rising blood nitrogen level lead to a fatal termination from renal failure. These authors describe a typical case illustrating these points, the patient dying on the fifth day. Small amounts of highly blood-stained alkaline urine, containing about 1% protein, were present, and nitrogen retention contrasted with depletion of blood protein. The alkali reserve of the blood was lowered. It has been generally assumed that the renal damage is caused by toxic material formed in the traumatized limb. On this hypothesis, various treatments, such as amputation, saline venoclysis, administration of suprarenal cortical extract, have been suggested. Patey and Robertson, subscribing to Blalock's theory of the ætiology of shock, consider that the kidney damage may be the result of dehydration from loss of fluids into the injured limb. Rather than raise the blood volume by intravenous injection, of fluids, therefore, they determined to try the effect of intermittently preventing the entry of blood to the crushed extremities. To this end they have employed the Paevex apparatus, originally introduced to assist a peripheral circulatory failure by enclosing the limb in a glass envelope within which the air pressure was phasically raised and lowered. The sheath proving unsatisfactory, they connected the Paevex motor and pump to a sphygmomanometer cuff of twice the normal size. Intermittent compression up to 50 millimetres of mercury was continued throughout the patient's waking hours, without increasing kidney damage, such as would be expected if the apparatus assisted in squeezing toxic bodies into the general circulation.

In two cases in which this treatment was followed, urinary output increased, a softening and then a gradual disappearance of the local œdema occurred, a fall in the blood urea level took place, and symptomatic improvement followed. This treatment, based on an entirely different concept of pathogenesis, is certainly worthy of further trial. In fairness, the writers point out that each patient on whom they experimented was not critically ill, and that the improvement might have been equally rapid without limb compression.

C. J. Longland and J. Murray[2] have recently described recovery in a severer case than the foregoing. Their patient's blood urea level climbed to 364 milligrammes *per centum* in five days with oliguria, blood casts and protein in the urine. The pelvis was fractured, and there was extensive bruising of the right side of the trunk and thigh as the result of burial under debris for four hours. These authors assumed that the renal damage was the result of hæmolysis from the release of a tissue hæmolysin by the injury. This view is supported by the fact that jaundice developed and the blood hæmoglobin level fell to 50%. A lactic acid acidosis is known to occur in muscle compression and injury, so that the stage was set for an accumulation of acid hæmatin in the renal tubules, such as may follow after a mismatched blood transfusion. Accordingly they treated their patient with normal saline solution given intravenously, followed later by 16 ounces of 3% sodium sulphate solution, coupled with generous doses of saline solution by mouth. Potassium citrate was administered from the third day, but the urine remained acid until the sixth day, when 360 grains of the salt were given. The blood urea level only started to fall on the eighth day, and from this point recovery was rapid. Longland and Murray make the interesting suggestion that the syndrome may be annulled if eight grammes of sodium citrate can be injected intravenously before the injured part is subjected to much disturbance.

These two views are not mutually exclusive, and each case would have to be judged separately. Obviously if the trunk and buttocks were severely traumatized, the Paevex machine could be of no service. If the limbs were involved, an inflatable band could be placed proximally around each, and perhaps be operated from the same motor. This would have the double effect of preventing fluid loss into the injured tissue and limiting the entry of the hæmolysin and lactic acid into the general circulation. Until further results are available it would, however, be a dangerous policy to cease the parenteral administration of hypertonic fluid once the kidneys were failing.

[1] *The Lancet*, June 21, 1941.
[2] *Ibidem*, August 9, 1941.

## MINERAL OIL.

IT is astonishing that in a country where natural food is good, plentiful and cheap, the population should have resorted so largely to all kinds of artificially prepared foodstuffs. If someone analysed how much money was spent on such preparations, from breakfast cereals to special beverages, he would arrive at figures that would probably be surpassed only by the amount spent in ridding the body of these precious additions to the daily menu. The habit of taking mild aperients, especially in the form of mineral oils, for all kinds of mostly imaginary complaints, has grown to an enormous extent; but it has always been thought that such a habit, while certainly beneficial to the manufacturers, was at least not detrimental to the consumer. It was generally believed that these oils could not be absorbed from the bowel and acted merely as a kind of lubricating agent, a thought very appealing to our motor-conscious age. But even this belief has been shaken by recent investigations of W. A. Stryker.[1] In rabbits, guinea-pigs and rats fed for months on a diet containing mineral oil, he found the oil taken up by phagocytes in the intestinal mucosa, mesenteric lymph nodes, liver and spleen. Chemical analysis showed that the oil seen in these phagocytes was actually the mineral oil that had been given to the animals. By examining the organs in a small series of cases *post mortem*, Stryker was able to show that similar absorption occurred in human beings. The tissue reaction towards the absorption of the oil has been of the type of foreign body reaction characterized by the presence of cells of chronic inflammation, including giant cells.

Pathological and physiological effects attributable to the deposition of oil in the tissues have not yet been demonstrated. It seems to be necessary for these investigations to be repeated on a much larger scale before it can be decided whether or not the use of such mineral oils over a period of years might lead to pathological changes. However, the results of the investigations add another strong argument against the indiscriminate use of such oils.

[1] *Archives of Pathology*, June, 1941.

# Abstracts from Medical Literature.

## GYNÆCOLOGY.

### Inoculations of Intestinal and Vaginal Trichomonads in the Human Vagina.

L. G. FEO, A. E. RAKOFF AND R. M. STABLER (*American Journal of Obstetrics and Gynecology*, August, 1941) present evidence that the intestinal trichomonas is unable to establish itself in the human vagina, even under conditions which are favourable to *Trichomonas vaginalis*. This shows that contamination from the rectum is not a means by which vaginal trichomonas is acquired. On the other hand, *Trichomonas vaginalis* could be transmitted from one patient to another; however, of the 25 patients infected, only two developed typical symptoms. The test was carried out on fifty volunteer patients. It was noticed that some of the patients who were infected with *Trichomonas hominis* had clinical symptoms of trichomonas although the authors were unable to detect *Trichomonas hominis* in the vagina.

### Intraperitoneal Hæmorrhage from the Ovary.

T. R. HOFFMANN (*The Western Journal of Surgery, Obstetrics and Gynecology*, June, 1941) presents a clinical study of 55 patients who suffered from intraperitoneal hæmorrhage due to rupture of a Graafian follicle or of *corpus luteum* cysts. Thirty-six of these patients were operated on and in 19 instances the patients were kept under observation and not submitted to operation. The author states that one of his objects is to draw attention to the fact that operation is not always necessary and that, even when operation is performed, ovarian tissue must not be needlessly sacrificed. He shows that 18 of the 36 patients need not have been treated by operation; in only eight of the 36 cases was a correct diagnosis made. In 17 cases the diagnosis was acute appendicitis, in five recurrent or subacute appendicitis, in six ruptured ectopic pregnancy, in two acute salpingo-oophoritis, in two acute mesenteric adenitis, in one pelvic abscess, and in one torsion of an ovarian cyst. If there is reasonable doubt of the correctness of the diagnosis, it is advisable to perform an exploratory laparotomy. The actual treatment of a ruptured cyst will depend upon the extent of the ovarian damage. This may vary from a minute stigma to what is virtually complete destruction.

### Gonorrhœa in the Female.

E. N. EAST AND S. A. MCFETRIDGE (*The Canadian Medical Association Journal*, September, 1941), working in the Division of Venereal Disease Control of the British Columbia Board of Health, have made a study of 1,712 women, both married and single, who presented themselves for examination. Of the whole group 626 were proved to have gonorrhœal infections. The majority of the women were either on relief or of low economic status. Their ages varied from 16 to 69 years, the average age being 22·9 years. They included women who sought advice on account of vaginal discharge, women who were brought for investigation as alleged sources of infection, and women who were referred by the law enforcement authorities for examination. A routine examination was made in all cases and extended over three days. On the first day smears were taken and examined by Gram's stain. If no evidence of gonorrhœa was found the urethra was painted with 2% silver nitrate solution and the cervix with a 5% solution. On the second day smears were again taken and the painting with silver nitrate was repeated. On the third day smears were taken and media were inoculated for incubation. *Trichomonas vaginalis* infection was present in 20% of cases. Gonorrhœal infection of the rectum was present in 8%, but only those patients who complained of rectal symptoms were examined. Infections were classified as acute, subacute and chronic. In the acute stage, local and surgical treatment are contraindicated; in the subacute stage surgical treatment is contraindicated, but may be undertaken in the chronic stage. All patients received sulphanilamide therapy. At least one course was given; if the drug was well tolerated, two courses were given. Pelvic complications were present in 9·2% of cases. Sulphanilamide tends to prevent complications not only because of its curative virtue, but also because during the acute stage, when local interference is contraindicated, it is generally used alone. Rest in bed during the menstrual periods, particularly the first, is important in the prevention of pelvic complications. The average duration of treatment in the acute cases was 18·3 weeks, and in the chronic cases 20 weeks.

### Inguinal Ectopia of the Ovary and Fallopian Tube.

V. MEYER AND F. G. TEMPLETON (*Archives of Surgery*, September, 1941) report a case of inguinal ectopia of the ovary and Fallopian tube, occurring in a girl, aged four months, and review 195 cases from the literature. A distinction is drawn between inguinal hernia of the ovary and inguinal ectopia of the ovary. In the former the ovary alone is found lying in the inguinal canal, while in the latter the ovary is accompanied by the Fallopian tube. In both conditions there is a persistence of the canal of Nuck. The production of inguinal ectopia of the ovary is dependent on anomalous development of the genital tract. In the authors' opinion the presence of the ovary and Fallopian tube in the inguinal canal represents a full descent during fœtal development, and corresponds to descent of the testicle from an intra-abdominal to a scrotal position. They point out at the same time that Watson does not agree with this view because the testicle is retroperitoneal and the ovary is intraabdominal and has a free mesentery. In the case reported by the authors operation became necessary when the patient was nine months old. An incision of the Bassini type was made, and when the sac was opened the right ovary and Fallopian tube were found to be deeply cyanotic and moderately œdematous and attached to the labium by a short gubernaculum of the ovary. They were freed and returned to the abdominal cavity. The operation was completed and recovery was uneventful. From their review of the literature the authors cannot agree that the condition "is more common in the presence of a bifid uterus". Sometimes resection of the ovary and tube is necessary, but both structures should be preserved if possible, even though it is possible that the tube may become the site of an ectopic pregnancy in the child-bearing period.

### Œstrogen and the Menopause.

S. M. R. REYNOLDS *et alii* (*Surgery, Gynecology and Obstetrics*, August, 1941) write on the dermo-vascular effects of œstrogen in women suffering from menopausal flushes. When œstrogen is given by injection to women in the menopause the vasodilating effect on the small blood vessels of the skin is of two kinds. One effect is manifested by a slow progressive change. It may be measured by a finger plethysmograph; the finger increases in size. This is called the plateau response. While the plateau response is developing a sudden increase in finger volume may occur which lasts for from three to fifteen minutes and subsides to a point somewhat above the preinjection level. This is called the flush type of response. The relative frequency of these responses in women in the menopause is affected by the clinical condition of the patient at the time of the injection. Before the menopausal symptoms are relieved the flush type of response predominates; after clinical relief the flush type of response diminishes in frequency and the plateau response increases somewhat. (In a certain number of cases no response to the œstrogen is demonstrable.) The changes in the relative frequency of the types of response were found by the authors to be most pronounced in women whose menopausal condition was relieved through removal of a complicating neurosis and least pronounced in those in whom no psychogenic factor was discovered. The authors conclude that clinical improvement in the vascular disturbances of the menopause brings about certain stabilizing adjustments on the peripheral vascular tree, affecting both the arterioles of the skin and the capillary blood vessels.

## OBSTETRICS.

### Ocular Complications in Hyperemesis Gravidarum.

A. J. BALLANTYNE (*The Journal of Obstetrics and Gynæcology of the British Empire*, April, 1941) points out that ocular complications in *hyperemesis gravidarum* are far more common than is thought, and that the first symptom is probably a retrobulbar neuritis, which, like other nerve involvement in pregnancy, is associated with vitamin $B_1$ deficiency. The latest stage is retinal hæmorrhage. Although the patients may have total blindness at the time, after induction they may recover almost complete vision. In the author's opinion an early examination of the *fundus oculi* would help the clinician to decide whether termination and the induction of labour are indicated. Cases are quoted to show that the administration of vitamin *B* improves the prognosis and may prevent a fatal termination of the condition. The author also expresses doubt regarding the purely neurotic element in *hyperemesis gravidarum*. The active ocular changes are first a retrobulbar optic neuritis associated with a central defect in the field and without visible ophthalmoscopic changes. This is succeeded by a visible

œdema of the optic nerve, and lastly by a characteristic type of peripapillary retinal hæmorrhage. The ultimate visual defect may amount to total or almost total bindness, which, however, is capable of complete cure.

### Sacral Anæsthesia in Obstetrics.

W. H. POOLE (*The Journal of Obstetrics and Gynæcology of the British Empire*, February, 1941) has adapted a technique of sacral anæsthesia for use in obstetrics. The method was successful in over two-thirds of the cases. There were no complications. "Percaine", 25 to 45 cubic centimetres of a 0·1% solution in normal saline solution, was used. Anæsthesia was sufficient for internal manual manipulations and suture of the perineum, but was insufficient for delivery by forceps. Anæsthesia lasted four hours. This form of anæsthesia should not be used unless the contractions are strong and regular. Obesity is a contraindication.

### Albuminuria of Pregnancy.

J. ST. G. WILSON (*The Journal of Obstetrics and Gynæcology of the British Empire*, April, 1941) gives the results of the treatment of 900 patients suffering from all types of albuminuria of pregnancy. Treatment in the various stages of the condition is discussed with detailed treatment of the 63 patients suffering from eclampsia with two deaths. The author endeavours to establish a balance between fluid intake and output, and the patients are put on a restricted water intake. Twenty ounces are arbitrarily decided upon as the minimum amount of intake; if the output is less than this, it is an indication that induction should be carried out. The author also believes that the failure to establish a water balance is more serious than the rise of blood pressure. He is an advocate of the intravenous administration of 20% magnesium sulphate; ten cubic centimetres are injected intravenously, and it is the author's custom to inject with it ten cubic centimetres of calcium gluconate to counteract the respiratory depression. This is used daily, and if no improvement has taken place within seven days induction is advised.

### A Study of Twin Pregnancies in the Argentine.

GERMÁN ROBERTO WERNICKE (*Revista Médica Latino-Americana*, May, 1941) surveys a series of 353 Argentine women and 318 foreign women having twin pregnancies. Heredity is an important factor; in 20·60% there was a familial history of twins, on either the maternal or paternal side; 2·08% of the women had second twin pregnancies. Consanguinity of the parents may be an influencing factor. Syphilis plays a minor role; it was demonstrated clinically and serologically in 1·31% of the mothers (although 7·46% had suspicious histories). The author considers that multiple pregnancy is pathological, since its complications — hydramnios, abortion and premature labour—are of the "gravid-toxic" order. Complications, such as eclampsia (0·89%), *placenta prævia* (0·76%), prolapse of the cord *et cetera*, obscure the prognosis. Among all women in the Buenos Aires district twin pregnancies bear to single pregnancies the ratio of 1 to 86; triplets occur once to every 4,828 single pregnancies; purely Argentine women have the ratios 1 to 96 and 1 to 4,877 respectively. Among Spanish women in the Argentine twin pregnancies occur once to every 62 single pregnancies. Of all the multiple pregnancies, 25·48% are univitelline; among Argentine women the incidence is 27·55%. There is a high mortality (21·31%); univitelline multiple pregnancies have a higher mortality (29·65%) than bivitelline (18·40%). The principal causes of mortality are: congenital debility, 8·0%; prematurity, 3·87%; bronchopneumonia, 1·19%; macerated fœtus, 2·67%; and the remainder are due to malformations and trauma during delivery. Of the fœtuses of univitelline pregnancy, 6·68% are born in the macerated state. Twins born of Argentine mothers have a death rate of 22·04%. The average weight at birth is 2,461 grammes; in the bivitelline group the weight is 2,500 grammes, and in the univitelline 2,250 grammes. The first child born is usually the heavier, the average weights being 2,441 grammes and 2,363 grammes respectively. Twins of Argentine mothers delivered at term averaged in weight 2,695 grammes (bivitelline) and 2,538 grammes (univitelline); those of foreign mothers averaged 2,743 grammes and 2,587 grammes respectively. The sex distribution was: two males, 33·47%; two females, 43·72%; male and female, 30·55%; 60% were of the same sex in bivitelline pregnancies. There were 100 males to 104 females among the twins born of Argentine mothers. Malformations occurred in 2·16% of cases. Operative interference was necessary in 16·15% of the deliveries. Only 30·70% of multiple pregnancies end in the spontaneous delivery of live babies of good weight. Ninety-two children from twin pregnancies were examined at the end of the first year; 54·28% were below normal weight, 8·69 had hernia, rhachitic signs were present in 48·21% of Argentine parentage and in 58·33% of foreign parentage. Only 34·79% of the children could be considered healthy.

### "Uroselectan B" as an Agent for the Induction of Labour.

P. LYON PLAYFAIR (*The Journal of Obstetrics and Gynæcology of the British Empire*, February, 1941) reports a series of 115 inductions of labour by the intrauterine injection of ten cubic centimetres of "Uroselectan B" through the abdominal wall. In four cases regular labour pains had not commenced within seventy-six hours of the injection. In these four cases the fœtus was alive before the injection and died either before or during labour. The author estimates that the fœtal mortality directly attributable to the method of induction was 5·2%. It is suggested that this method may prove useful in certain types of case. The success rate was 96·25% and no deleterious effect was observed in the mother. If "Uroselectan" is injected directly into the maternal or fœtal tissues it may cause considerable sloughing.

### Hepatic Hæmorrhage in Stillborn and Newborn Infants.

J. L. HENDERSON (*The Journal of Obstetrics and Gynæcology of the British Empire*, June, 1941) found 47 cases of hepatic hæmorrhage in a series of 1,312 post-mortem examinations on stillborn and newborn infants. Its incidence was three times as great in stillborn infants as in those who died within one week. There was an associated intracranial hæmorrhage in twenty cases. Infants of average weight suffered less than premature or large infants. In only three cases was delivery spontaneous. Complicated labour was the chief predisposing cause. In the infants that survive birth, diagnosis is seldom made during life. The sudden appearance of a severe anæmia during the first few days of life should lead the clinician to suspect the condition. *Paracentesis abdominis* should then be performed to confirm the diagnosis. Treatment should be by surgical intervention after transfusion. In three-quarters of the cases the hæmorrhage was situated on a surface subjacent to the abdominal or thoracic wall. The administration of vitamin *K* may be tried.

### Pelvic Delivery under Local Infiltration Anæsthesia.

CHARLES P. SHELDON (*The New England Journal of Medicine*, March 6, 1941) believes that small doses of sedatives during labour, in addition to local anæsthesia for delivery, give the most satisfactory results in normal and low forceps deliveries. In addition, by these means, spontaneous breech deliveries, manual rotation of the head, Scanzoni's operation, delivery in face and brow presentations, and even mid-forceps deliveries can often be conducted under local infiltration anæsthesia. The procedure is simple and safe. The author uses 50 to 70 cubic centimetres of a 1% solution of "Novocain" with adrenaline to block the nerves. Morphine, hyoscine and "Seconal" are administered. Nitrous oxide and oxygen are given while the perineum is being prepared and infiltrated. If delivery is not progressing rapidly, an episiotomy is performed and the forceps are applied. The author claims that the anæsthesia lasts for one and a half to two hours, muscular relaxation is produced, blood loss is slight, and the condition of the mothers and babies is well maintained.

### Prophylactic External Cephalic Version.

J. L. NEWELL (*American Journal of Obstetrics and Gynecology*, August, 1941) records the results of the practice of external cephalic version at the Boston Lying-In Hospital for a period of four years. This survey covered 13,948 patients. A diagnosis of breech presentation was made in 793 cases; 1,161 versions, or 1·46 per case, were attempted, 72% being successful. Spontaneous version occurred in 19·35% of cases in which recurrence had taken place or attempted version had been unsuccessful. Anæsthesia was employed only in selected cases. During the period under review 246 breech deliveries occurred, and in 60% of these there had been no attempt at version, as the diagnosis was not made until the patient was admitted to hospital for delivery. However, the incidence of breech delivery was reduced from 3·75% to 2·33% for the previous four years. The greatest number of successful versions were performed at the thirty-second week; from this on they became progressively less successful. In only one of the 1,161 versions was death due to the cord being round the neck, and one other death was attributed to a true knot. There were five macerated fœtuses, but all had fœtal heart sounds one week after version. The author concludes that external cephalic version employed as a routine measure is safe and will reduce by more than 38% the incidence of breech presentation, with its inevitable high fœtal mortality.

# British Medical Association News.

## SCIENTIFIC.

A MEETING of the New South Wales Branch of the British Medical Association was held on August 21, 1941, at Broughton Hall Psychiatric Clinic. The meeting took the form of a number of clinical demonstrations by members of the staff. Part of this report appeared in the issue of November 8, 1941.

### Presbyophrenia.

A male patient, aged forty-nine years, suffering from presbyophrenia, was shown; he was a sergeant of police, married, with five children. He had been admitted to Broughton Hall on February 12, 1941, with a history of advancing loss of memory for recent affairs. His father and brother had died of apoplexy and his mother of renal disease. The patient's early life was uneventful; he had been for many years in the police force, and was in charge of a police district in the country. He rarely took alcohol and was a light smoker. He had had dengue fever, and pneumonic influenza in 1919; he suffered from chronic rheumatism, and in 1929 he had to use crutches. For about a month prior to his admission to hospital he had had partial failure of memory for recent events. He tried to conduct the same court cases twice, forgot the reasons for which people paid him money, fines *et cetera*, and where he had put his belongings, and became unable to perform his routine police station duties.

On his admission to Broughton Hall he exhibited confusion, loss of recent memory for many things, poorness of concentration, loss of interest and slowness of speech with inhibition. Since then he had become hallucinated for voices and vision, and had had delusions of a persecutory nature, on one occasion assaulting the patient in the next bed. He had become more confused and showed further deterioration; he had Korsakow's type of confusion and was restless. He had delusions of a bizarre and rapidly changing kind; he thought people shot bullets at him in streams, and that the town was on fire and he put out the blaze. On some days he conducted his routine court work in the single room. At times he had shown negativism and aggression, but for the most part he was friendly to the staff.

The patient was tall and well built. The systolic blood pressure was 144 millimetres of mercury and the diastolic 78; the vessels were healthy. The knee jerks were equally exaggerated on both sides, and pronounced continuous tremor of both hands was present, which was becoming coarser. He vomited several times and complained of pain in the occipital region. Examination of the optic disks revealed no abnormality. In view of the possibility of a cerebral tumour, the patient was sent to the Royal Prince Alfred Hospital, where Dr. Gilbert Phillips examined him. Dr. Phillips reported that the pressure of the cerebro-spinal fluid was low (40 millimetres) and that the protein content of the fluid was not increased; an encephalogram revealed very little air in the ventricles, but there was a considerable amount over the brain surface; the depth of this air showed that the patient had extensive cerebral atrophy, the brain in some places being two centimetres away from the inner table of the skull. Dr. Phillips considered that the findings excluded the possibility of cerebral tumour. Complete serological examination produced only negative results. The patient's mental deterioration had rapidly progressed. It was pointed out that, although he had previously been a good shot with a sporting gun, he had given up that sport a few years earlier because of a tremor of his hands.

The comment was made that the patient's clinical history illustrated the group of presenile dementias that included Alzheimer's disease and Hicks's disease. Striking features were the insidious onset and the extremely rapid progress. It had been possible to observe in the case under discussion the rapid deterioration of the mental faculties over a period of a few months from a state of good efficiency to one of almost complete dementia. At the time of the meeting the man had no power of voluntary speech, although sometimes he exhibited reflex or automatic speech. For example, if someone shook hands with him, sometimes he would say: "How are you?" The pathological condition underlying the dementia was a progressive atrophy of the cortex, with numerous areas of sclerosis consisting of the so-called senile plaques.

### Multiple Sclerosis.

A female patient, aged twenty-five years, suffering from multiple sclerosis, was shown; the patient was married and had one child. Her father had died recently from lead poisoning. The patient had lived all her life at Broken Hill; she had had measles, pneumonia and catarrhal jaundice. For ten weeks before the birth of her baby in August, 1937, she had been treated in hospital for albuminuria. Labour was induced because of preeclamptic toxæmia. She remained in hospital for six weeks after her confinement, and then returned home, well, except for stiffness in her left leg. She was treated for this condition, but showed no improvement, and soon afterwards her right leg became affected. Her condition had become gradually worse, so that at the time of the meeting she was able to walk only one or two steps with assistance. During the last year her vision had become blurred; her speech had been indistinct during the last eighteen months.

The patient was unable to walk or stand without much assistance; both hands and legs were grossly ataxic. Tremor of the hands and head was present, and the speech was slow and indistinct. Slow nystagmus, both horizontal and vertical, was present. Her vision was blurred, more so in the right eye than in the left. Both optic disks were pale; the pupils were small and irregular and reacted to light, but she had difficulty with accommodation. She became giddy on sudden movement of her head. The leg muscles were flabby. The knee jerks were exaggerated and jerky on both sides; the plantar reflex on both sides was extensor in type; the Achilles reflex was present; the wrist reflex was present on the right side and exaggerated on the left; ankle clonus was present on the left side. The patient had precipitate micturition, with slight control over the bladder and rectal sphincters. Pain sensation was normal; temperature sensation was impaired on the anterior aspect of the legs; muscle, joint and tendon sensation was impaired. The patient had a persistent cough with sputum; X-ray examination revealed no pulmonary lesion. She had been treated with *Mistura Quininæ Sulphatis*, five grains three times a day, and by a diet of liver, brains, "Marmite" and orange juice. Massage and attempts at muscle reeducation were also used. It was pointed out that the patient's condition illustrated the not uncommon experience of finding multiple sclerosis dating from a pregnancy. The course had been rapidly progressive and she had had no remissions; the prognosis was grave.

### Disseminated Sclerosis.

A male patient was shown, aged twenty-seven years; he was unmarried and was a radio technician by profession. He had been admitted to Broughton Hall on July 4, 1940. His father was a heavy alcoholic. The patient had had pneumonia and pleurisy three times and severe influenza about six years earlier. He had been of normal disposition until about eighteen months previously, when he became quiet and indifferent, lost his interests and began to sit idly about the house in his spare time. He was on night work, from 10 p.m. until 8 a.m., and this did not suit him; he became depressed, and at Christmas, 1939, he was dismissed. While going to church he noticed that his legs were tired and he stumbled; he was hardly able to reach his destination. At the table he began to drop his knife and fork and to spill food. He was naturally left-handed, but wrote with his right hand, and his penmanship became poor as the weakness of his hand increased. He could not speak properly; his tone became unemotional and monotonous, and he had difficulty with conversation, as his speech muscles became easily fatigued. He gave up reading because of the presence of diplopia, and horizontal nystagmus developed. At times he would have fallen but for assistance. He ate well, slept too deeply and was very "touchy". The diplopia caused him to walk into obstacles and to stumble. In December, 1939, he was examined for the presence of a cerebral tumour, with negative results. Serological investigation revealed no abnormality. In 1940 his condition rapidly became worse; his gait was ataxic and he could not use his hands properly to stir tea, write or perform any of the usual movements, owing to increasing enfeeblement of motor power and tremor. The diplopia became worse and objects appeared to travel upwards and to the right when he looked at them. Dizzy spells developed later; he used to fall, but remained conscious. He dragged his right leg; the speech disorder had considerably increased, and he often said the wrong words.

He was given a course of injections of "T.A.B." vaccine, but no benefit resulted. In the middle of 1940 he was given a course of inductothermy. He felt that his head was clearer after each session, but later he retrogressed. In November, 1940, he was discharged from hospital; his mental condition was much better, but otherwise he was not particularly improved. He was also given thyreoid gland treatment, small doses being used. He was readmitted to Broughton Hall on March 12, 1941; he was cheerful and cooperative, and his physical signs showed slight remission. Inductotherm treatment was again instituted.

At the time of the meeting the patient felt well, but his condition was stationary. He was, however, less troubled

by the diplopia. Horizontal and vertical nystagmus was present in both eyes, some weakness of the left internal rectus muscle was seen, muscle tone was slightly less in the right leg and arm, and he dragged the right foot. He showed adiodokokinesis and some incoordination of movements; he tended to fall to the right when he turned to the right, and Rombergism to the right side was present. He had intention tremors, and the deep reflexes were exaggerated. He had lately begun to suffer from pain over the right *quadriceps femoris* muscle, worse at night. A course of inductotherm treatment lasted for 100 hours, and the maximum temperature attained was 105° F.

It was pointed out that the treatment of disseminated sclerosis was not yet satisfactory. Heat treatment had been tried with some good and some bad results. Subjectively, the patient under discussion felt that his condition was improved; objectively, probably he was very little better.

### Congenital Cerebral Palsy.

A female patient, aged seventeen years, suffering from congenital cerebral palsy, was shown. The patient was unmarried and was the eldest of five children; all the others and the parents were healthy. The mother had had a difficult labour and instrumental delivery was necessary; the patient was paralysed on the right side from birth. At the age of five months the patient was treated at a hospital, where her right arm was put in a splint. She was late in starting to walk and talk; she did not put her heel to the ground when attempting to walk. No operation was performed. Later an operation (cervical ramisection) was performed for her arm. She was a backward child, and learned to write only with the greatest difficulty. At the age of thirteen years she began to have major epileptic convulsive seizures and was taken from school, as she had some serious falls. Since then she had done nothing except a little housework. During the last two years she had been flying into rages if opposed. She was uncontrollable at home and went out against the parents' wishes; she persisted in frequenting the company of men whom the parents believed to be undesirable. She appeared to have no moral sense. She had attended for some years the hospital where her right arm was originally treated, and later attended another hospital. It was found that "Dilantin" tablets controlled her better than "Luminal", bromides *et cetera*. Since her admission to Broughton Hall she had been quiet and amenable, although she admitted to being irritable, quarrelsome, impulsive, self-willed and sullen at home. Mentally she was retarded, her intelligence quotient being 67·2% and her mental age ten years and nine months.

The patient presented a hemiparesis of the right arm and leg; the right side of the face was asymmetrical, but not much paresis was noted in that area. She had hypertonia of the right arm and leg and to a less degree of the left leg. She exhibited reflex grasping of the right hand, but could not voluntarily grasp with that hand. She dragged her right leg, but did not swing it. Her right arm was held in an attitude of hyperextension, with hyperflexion at the wrist and alternating hyperextension and hyperflexion of the fingers. Occasional choreo-athetoid movements were noticed. The knee jerks were exaggerated on both sides; the ankle jerk was present on both sides; the wrist reflex was exaggerated on the right side and was present on the left; clonus was elicited at both knees and at the right ankle; it was not elicited at the left ankle. Pain sensation was exaggerated at the right foot and leg (thalamic response). The pupils were unequal, but reacted to light and accommodation. No nystagmus was present. The visual fields were restricted, but no gross abnormality was detected.

It was remarked that an encephalographic examination of the patient revealed extensive porencephaly. Neurologically the interesting feature of the case was the tendency for the toes spontaneously to assume the attitude of extensor response.

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A MEETING of the New South Wales Branch of the British Medical Association was held on July 24, 1941, at the Women's Hospital. The meeting took the form of a number of clinical demonstrations by members of the honorary medical staff of the hospital.

### An Exhibit of Cannulas.

DR. ALAN GRANT presented an exhibit of cannula curettes used to obtain specimens of endometrium for biopsy, no anæsthetic being necessary. A printed list of the uses of endometrial biopsy accompanied the exhibit and stressed its importance in the diagnosis of anovulatory menstruation and functional uterine hæmorrhage.

There was also an exhibit of cervical cannulas used to inject iodized oil into the uterus and Fallopian tubes, including one which obviated the use of a volsellum.

### Congenital Dextroversion of the Uterus.

DR. S. D. MEARES showed an X-ray film and diagrams of a patient who had been shown at a clinical meeting at Sydney Hospital on September 19, 1940, with a gestation in a bicornuate uterus. The patient had since had a normal labour and puerperium. Recently Dr. Meares had injected lipiodol into the uterus and taken an X-ray picture. It was thereby revealed that the uterus was single and dextroverted and the Fallopian tubes were normal. The main interest in the case lay in the fact that the diagnosis of a congenital abnormality was now changed to that of a congenital dextroverted uterus with septate vagina. Dr. Meares pointed out that occasionally complete or partial duplication of the vagina occurred with a single uterus, but in most cases the uterus also was double.

### The Fertility Assay of Spermatozoa.

DR. R. MACKEY discussed the fertility assay of spermatozoa. He said that a great deal of diffidence on the part of many medical men had led to neglect of the examination of human semen as a routine measure in the investigation of sterility. When it was considered that 20% of childless marriages were due to deficiencies in the spermatozoa, the importance of that examination need not be stressed further. Nor was absolute sterility the only occasion on which the sperm cells could be at fault, for clinical and statistical evidence now pointed to the sperm cell as a major factor in the production of repeated miscarriages, monsters and hydatidiform mole. Further confirmation could come only by repeated examinations, and these did not entail much extra work.

Referring to the method of collection of the specimen, Dr. Mackey said that the condom method had proved itself satisfactory. Immediately after intercourse the contents were transferred to a clean but old (non-alkaline) glass tube which was provided. The best method was for the patient to cut the end of the condom with scissors. The tube was then corked and brought for examination, the patient being warned not to cool or heat the specimen. The viscosity was noted; the specimen should be fluid after one hour. The volume was normally between two and four cubic centimetres; amounts below one cubic centimetre were regarded as abnormal. A drop of semen was examined under a coverslip with a high-power lens; over 70% of spermatozoa should be motile in a normal specimen. Abnormal and immature forms were often visible in the wet specimen, as well as other cells—organisms, granules and crystals. Motility might persist up to thirty-six hours at room temperature; the average life under a coverslip seemed to be from six to eight hours. A dilution of semen to one in ten in a 1% solution of chlorazine in distilled water killed the sperm cells, and by making the mucus soluble produced a more even suspension. A standard hæmocytometer counting chamber was used for the actual count. The normal figure lay between 100,000,000 and 150,000,000 sperm cells per specimen. If less than 50,000,000 were present the specimen was regarded as abnormal.

Dr. Mackey then discussed morphological abnormalities. He said that Moench had stated that the sperm head was the greatest single source of information as to the fitness of the cells for reproduction, and in average cases the relative number of abnormal sperm heads afforded an index to the reproductive fitness of the subject. Yet this was the most persistently neglected part of any sperm cell examination, and it was of much more importance than a differential cell count of the blood. The easiest method was to make a smear on a clean slide from a fluid part of the semen and fix it with heat. The smear was stained with Wright's stain for four minutes, then diluted with an equal volume of tap water. It was left for two minutes, washed, dried and examined under an oil immersion lens. In normal semen the abnormal sperm heads did not exceed 19% or 20%. When the sperm head abnormalities reached 20% to 23% impaired fertility could be assumed, and when the figure was above 25% clinical sterility was usually present. Abnormalities included deformed heads, such as microforms, or round, tapering, asymmetrical or double heads, or absence of heads. The body might be deformed and the tails duplicated or deformed. Remnants of epithelial cells were occasionally seen adherent to the heads, and frank epithelial forms were occasionally seen. When aspermia was suspected the specimen should be centrifuged at high speed and the deposit examined. A differential cell count should be made on at least 200 sperm cells.

Dr. Mackey went on to say that the presence of starch-like granules and excess crystals was generally an adverse factor. Pus did not appear to interfere with motility or

fertilizing ability. Organisms were occasionally recovered. The main factor in the assay of fertility was the sperm head; this he regarded as of prime importance. Absence of motility, a low sperm count or a low volume of semen were secondary factors. In assaying a specimen he classed it as fertile, subfertile or infertile; but in the last two classes repeated examinations were necessary, if possible after three days' abstention from intercourse. The difficulty of translating a statistical result into a biological fact was admitted, but the experience of observers, both in the sterility clinic at the Women's Hospital and in clinics abroad, had shown that at the present time the universal requirements for a fertile specimen that had been described were in accordance with clinical results.

### Breast Feeding.

DR. SELWYN HARRISON presented a coloured film illustrating various phases of breast feeding. Normal breast feeding was first shown with several types of breast and nipple, followed by the difficulties of lactation due to maternal and infantile causes, and their treatment.

Dr. Harrison considered that films designed for teaching purposes would play an increasingly important part in medical education.

## The Royal Australasian College of Physicians.

### MEETING IN MELBOURNE.

A MEETING of the Royal Australasian College of Physicians was held in Melbourne on Friday and Saturday, September 26 and 27, 1941. The meeting was well attended by Victorian Fellows and Members, and a number were also present from all other States. The meeting was held in the building of the Royal Australasian College of Surgeons, Spring Street, Melbourne.

A meeting of the council of the college was held on the evening of Friday, September 26. Those present were the President (Dr. S. V. Sewell), Sir Charles Backburn, Sir Trent Ch. de Crespigny, Dr. A. J. Collins, Dr. A. Holmes à Court, Dr. S. O. Cowen, Dr. O. A. Diethelm, Dr. Konrad Hiller, Dr. F. S. Hone, Dr. L. S. Latham, Dr. Alex. Murphy, Dr. F. J. Niall, Dr. H. J. Ritchie, Dr. M. D. Silberberg, Dr. S. A. Smith and Dr. A. H. Tebbutt. A meeting of the joint councils of the Royal Australasian College of Physicians and the Royal Australasian College of Surgeons was held on Saturday, September 27.

### Scientific Programme.

At the first scientific session E. Weston Hurst, D.Sc., M.D., F.R.C.P., of Adelaide, delivered the first G. E. Rennie Memorial Lecture. The subject of the lecture was "Demyelination: A Clinico-Pathological and Experimental Study". Dr. Weston Hurst opened his lecture by an appreciation of the late Dr. G. E. Rennie's distinguished academic career in Sydney and in London and of his contributions to medicine, and particularly to neurology, during his active career as a physician in Sydney. This lecture will be published in a subsequent issue.

The following contributions were also given: "Treatment of a Case of Pneumococcal Meningitis", by Dr. Guy Springthorpe (Melbourne); "Some Observations on the Effects of Selective Irradiation of the Stomach in Cases of Chronic Non-Obstructive Duodenal Ulcer with Hyperchlorhydria", by Dr. W. P. Holman (Launceston); "Dissecting Aneurysms", by Dr. M. C. Davis (Melbourne). A symposium on cerebro-spinal meningitis was held on Saturday, September 26, to which all members of the Victorian Branch of the British Medical Association were invited. Colonel M. J. Holmes, D.S.O., spoke on the epidemiology, Dr. F. M. Burnet on the bacteriology, Dr. S. O. Cowen, Dr. J. W. Grieve and Dr. S. A. Smith on the clinical aspects and treatment. A discussion, opened by Dr. F. V. G. Scholes, followed.

### Admission of Fellows.

Dr. R. S. Ellery (Melbourne), Professor D. H. K. Lee (Brisbane) and Professor Sydney Sunderland (Melbourne) were elected to Fellowship of the college at a meeting of the general body of Fellows held on September 27. The President admitted Dr. Ellery and Professor Sunderland in person to Fellowship.

## Correspondence.

### THE PSYCHIATRIC EXAMINATION OF RECRUITS.

SIR: With diffidence I make, of your courtesy, some comment on this interesting subject. The adjective is deliberate. With due deference, as befits "a child in these great matters", I propose that far too much fuss has been, is being, and—by all portents—will continue to be made on this subject of psychic instability. With a definite and reasonably assessable residue of human material that is irrevocably psychopathic, through nature or nurture, the significance, speaking broadly, of psychic instability is just what we make it. This (I propose) is true of any social circumstance or episode wherein men are subject in mass to special strains and stresses. It is emphatically true of war. Through a curious and intriguing concatenation of circumstances the first phase of this World War (1914-1918) was permitted to bring about what must be almost a world record in mass suggestion. Partly through the immaturity of psychological medicine, partly through mismanagement, but chiefly through the impact of a biological shock unprecedented in the history of mankind, the perfectly legitimate medical concept "shell-shock" (that is, the effect on a soldier's behaviour of close proximity to a high-explosive shell-burst) which was designed to define one factor in the problem of "nervous breakdown"—what's wrong with this phrase, except simplicity?—became a devastating fetish. *Omne ignotum pro magnifico*—especially when the "unknown" presents an unconsciously enticing escape to "peace with honour".

While it were foolish to minimize the importance of the mental factor in military wastage—is not "the moral to the physical as 3 to 1"?—it is also unnecessary to over-estimate it. In the Great War, when the mass effect of the terrific shock of the Somme had been met by "N.Y.D.N." mental breakdown became a quite manageable cause of wastage—infinitely less important than that produced by the itch-mite or the body louse. (But ask a journalist which counts in "news value", "shell-shock" or scabies!) In the Australian Imperial Force the total figures for evacuation from all forms of nervous breakdown on the Western Front, including "S.I.W." and "N.A.D.", was in the neighbourhood of 7,000 out of a total evacuation to field ambulance for non-battle casualties of 211,822—a matter of 3·3%. To these must be added some thousand diagnosed cases of "D.A.H." or "effort syndrome". Specific skin infestation was responsible for over 20,000 evacuations—and this takes no account of trench fever or "P.U.O." Pensions, of course, are another story, and not directly relevant to the present issue.

To come to this. The problem in the medical examination, as it seems to me, is threefold, namely, to ensure that men are not finally embarked for overseas who (a) are pre-ordained to discharge "without service", or with excessive "overhead" in training, and time spent in hospital; (b) are pre-destined to heavy pension claims without compensating offset in useful service; (c) will be called on to suffer through serving their country in the army to an extent that is out of proportion to the value of the service which they might render in other ways.

To promote these desiderata I suggest:

That while it is, apparently, impossible to provide for a probationary period the training period should, at least from the medical standpoint, be regarded as a period of probation. At the medical examinations—preliminary, or by board of two—obvious mental unfits and misfits should be detected; if necessary with the help of "experts". In particular, the consequence of giving false answers to the questions designed to disclose defects in family and personal history should be made clear to the recruit. And it may not be out of place to suggest that the medical profession should insist that the conditions under which the examinations are made are not unnecessarily crude or repulsive.

But the effective discrimination of the "neurotics" or even the "psychotics" should be a function chiefly of the R.M.O.'s in the training camps; acting, as always, in close cooperation with the combatant officers, commissioned and non-commissioned. The chief points are:

(1) That time and opportunity are available for observation impossible in the rush and tumble of recruiting.

(2) In a large proportion of enlistments temperamental unfitness for army life will become fairly obvious during the first few months of training. Of course the soldier's ability to stand the strain of battle can only be tested by trial; but (and this is my chief point) the nerveus breakdowns which are a cause of permanent wastage and source of pension claims are not chiefly those brought about by "shock". The "psychotic" is, *ab initio*, a "bad mixer".

(3) For the rest (to sum a vast problem in a phrase) the attitude adopted toward potential nervous breakdown should be the positive one of promoting (moral) health rather than the negative one of the prevention and treatment of psychotic disorder.

It is not necessary to enlarge on the involvements of such a policy. Are they not written, for all who would read and be wise, in the report of the British War Office Commission on "Shell Shock"? And our official medical history of the war has quite a lot to say on the factors which promote mental and physical health. And lastly, when all has been said against the "neurotic", it is recorded with authority that the proportion of honours and rewards won by men who had been treated in hospital for some form of nervous breakdown was scarcely, if at all, less than that in the whole soldier community.

Yours, etc.,

A. G. BUTLER.

Victoria Barracks,
Paddington,
New South Wales.
October 30, 1941.

## COLOUR BLINDNESS.

SIR: H.G. saw me because he had been declared colour-blind when enlisting for the Royal Australian Air Force.

I found the following facts:

1. He plays snooker and never makes a mistake in the colour of the balls.
2. He can pick correctly all colours of book covers, flowers, trees, grass *et cetera*.
3. He can pick out wools of all colours required correctly.
4. He is absolutely red-green blind to "ISHIHARA" in daylight. For example, with number 4 he sees 2 distinctly, with number 5 he sees 21 distinctly. When electric light is substituted for daylight he cannot see the 2 and 21, but sees, not clearly but quite definitely, 5 and 74 as the normal should. Therefore he is only red-green blind to "ISHIHARA" in daylight, but is not red-green blind to daily work. I leave it to someone better able to explain.

Yours, etc.,

H. O. LETHBRIDGE.

Narrandera,
New South Wales,
October 31, 1941.

*Post Scriptum.*—He picks up his red tees with no difficulty and sees the red flag as a normal does at golf. A positive red-green blind cannot do either.—H.O.L.

## "GLUCOSE D" AND LOW BLOOD PRESSURE OF MIGRAINE.

SIR: In THE MEDICAL JOURNAL OF AUSTRALIA of November 1, 1941, Dr. Burton Bradley calls our attention to the fact that many cases of migraine are relieved by the exhibition of "Glucose D". He is apparently not aware that migraine is often the presenting symptom of a chronic condition of hypoglycæmia; this fact has been known for many years, but has received scant recognition in the literature, and thus has not reached the knowledge of many medical men.

I have seen a number of these cases in the last year, and hope to publish an account of them shortly. Briefly, I may now state that in these cases a characteristic migraine occurs when the patient is hungry—for example on waking in the morning or after exertion following a small meal. Investigation of the starving blood sugar often shows a reading as low as 0·04; other cases give a figure within normal limits, but a glucose tolerance test shows a characteristic low curve; therefore it is always necessary, when this syndrome is suspected, to have the glucose tolerance test done rather than a simple starving blood sugar estimation.

As Dr. Bradley notes, glucose often aborts an attack if taken early; but the best treatment is a high protein diet, a glucose drink on waking in the morning, and a warning against exertion on an empty stomach.

Dr. Marjory Thomas, of Manly, first brought this syndrome to my notice and has carried out the blood sugar estimation on my cases.

Yours, etc.,

CHAS. W. DUN.

"Elsmere",
Manly,
New South Wales.
November 1, 1941.

## A GENERAL MEDICAL SERVICE FOR AUSTRALIA.

SIR: Dr. F. L. Davies has called attention to a most important matter on page 515 of THE MEDICAL JOURNAL OF AUSTRALIA, November 1, 1941, where he is reported as saying that "The Federal Council did not know what the profession wanted . . ."

Unless the Federal Council is informed of the opinion of the profession, it will be severely handicapped in any activity on behalf of the members.

There are many difficulties in the way of obtaining such information, as was pointed out by the Queensland delegates at the meeting of the Federal Committee at Hobart in 1934.

The Queensland Branch sought to overcome these difficulties by the creation of a body akin to the Representative Body of the Parent Association. To anyone interested, the constitution of The Institution of Engineers will show how this body is able to ascertain the opinion of its members quite expeditiously.

In regard to the question of future community and hospital medical services that will adequately safeguard the health of the people in all parts of the Commonwealth, the medical profession should not be in too great a hurry to exchange a system that is by no means altogether bad for an unknown one. We should seek evolutionary rather than revolutionary methods.

As Dr. Colville pointed out, there are a great many people in the community who are enjoying the benefits of private medical practice. Dr. George Bell's remark (page 516) indicates that New South Wales practitioners have similar ideas. This does not imply that there is no room for improvement in our medical services. Our policy should be to assist governmental and semi-governmental bodies to make such improvements as are necessary. We shall not be able to consider our medical services sound until they are, as far as possible, satisfactory to the patients, the State and the profession.

The necessary improvements vary greatly from State to State and in various districts within each State. Our most pressing need is to know public requirements throughout Australia.

In conclusion, there are three matters of urgency:

1. To place on record the varying conditions of medical practice in Australia.
2. To hold a meeting of at least one hundred representatives of the profession for the purpose of informing the Federal Council of the desires of members of the profession as a whole.
3. To take steps to see that every effort be made to implement the Federal Council's resolution that there be no radical change in the present form of medical service during the war.

Yours, etc.,

E. S. MEYERS.

639, Sandgate Road,
Clayfield,
Queensland.
November 2, 1941.

SIR: There is much loose thinking on the future of medicine in Australia, and much loose talking and writing. The one is bad, the other worse. There are two main themes, whether the author be a Minister of State, nurtured in nationalization, a government medical officer who has never practised medicine, or merely an occasional correspondent in THE MEDICAL JOURNAL OF AUSTRALIA. The one is that the present organization of medicine must be superseded by a "Nationalized Health Service"; the other is that the extravagant statements of policy by various governmental authorities, often unrelated to the truths of the question, should be accepted as accomplishments of policy, that nationalization being inevitable, men of medicine should begin in a variety of ways and degrees to emulate the men of Vichy.

At the outset let there be no mistake as to the objective of modern medicine in Australia, organization and personnel. Medicine lives and works for the ultimate good of the individual patient and so for the collective good. That is expressed in the Hippocratic oath in the original charter of the British Medical Association and in the foundation document of the American Medical Association. It has been reviewed in age after age in British countries and in the United States of America by every leader of medicine; it is echoed every day in the thought and practice of ordinary men of medicine. If, then, the structure of medicine in Australia has failed to achieve that objective or if it is so inefficient that that objective cannot be achieved, it is right either that its present form should be replaced with one with the prospect of greater efficiency, if indeed such a form could be constructed, or that it should be enlarged so as

to fulfil needs already capable of demonstration or anticipation. For every conscientious doctor this is the foundation of the question. Granted the Hippocratic objective there must be the maximum possible efficiency. No doctor ever built a lasting success except with that idea in his very soul.

Has medicine then failed for efficiency? Have the Minister and the correspondents forgotten? Or have they merely failed to see? Australia has evolved a medical organization eminently suited in its main structural features to the geography of the country, and so generally efficient as to have evoked the praise of authorities of reputedly the most advanced countries of the world. Possibly that same geography has been a main stimulus to the development of efficiency. Medical personnel has necessarily been trained for the enterprise and judgement required by the responsibility of practice in centres which are still often remote in spite of modern transport. The Australian graduate has a high ranking. But if geography has had a strong hand in this, so has spontaneous demand of the Australian profession through its leaders and its teachers for an ever higher standard of medical education. Was this inefficiency, this which we doctors all know to be true?

And our hospitals? We have built up chains of great hospitals comparable with the most efficient in the world. They and the hundreds of district hospitals and base hospitals throughout the Commonwealth exist in virtue of the charity of private individuals, the public spirit of numbers of citizens, the subsidy of governments, and the advice, the foresight and the service of leaders and rank and file alike of the men of medicine. There is scarcely a major advance in hospital construction, equipment, organization or practice in Australia which has not had its genesis in the constructive mind of some practising doctor and its fulfilment in the pursuit by the profession generally of higher efficiency. Is that a sign of inefficiency?

What of the medical service to the outback, this service which has struck the imagination of people in other countries? The Flying Doctor Service—a splendid system that and, in face of all the difficulties, so efficient. Born of an idea from the mind of a great Christian, organized and directed with the knowledge, the enthusiasm and the wholly voluntary advice of able practising doctors and private citizens. A perfect example of that enterprise which is so much a quality of medicine in Australia. Could you speak of inefficiency in this?

There is much talk from official circles of preventive medicine? As though medicine has not been alive since the first days of the knowledge of preventive medicine to its surpassing importance, as though, as Dr. Willcocks so clearly pointed out in a recent letter, the profession had not been engaged every day for decades in practice on individuals of some form of prevention—asepsis in surgery and midwifery, immunization in smallpox, diphtheria, whooping cough and tetanus, the isolation of the infectious in tuberculosis and specific fevers—in these and in a hundred little ways we continue to extend the practice of preventive medicine. Moreover, organized medicine has led a public demand for it while governments have failed, as they fail now, for example, to take adequate measures against the spread of tuberculosis in breadwinners, even after they have been strongly pressed to do so by the Federal Council of the British Medical Association. The last organization to move is ever the government.

Then suddenly at length a Minister or a chief government medical officer awakes or they awake together, and with an air of quasi-sublime innocence they inveigh with suspicious unison: "You doctors who have initiated the movement in every department of preventive medicine, who have led and carried out the attack on maternal mortality, who show us now the means of controlling tuberculosis which we do not seize, you who with the wise assistance of the Australian Broadcasting Commission have pioneered popular medical education in Australia before hundreds of thousands of listeners, you who have taken a major part in increasing the average expectation of an Australian life by fifteen years in the forty years of this century, you doctors who wish to remain efficient, we are going to destroy you for inefficiency."

Very well, on that issue we doctors will not flinch from a conflict. I for one consider we should tell you that now.

Why do they seek to destroy an organization, full of enterprise and high purpose, built up over a century by splendid useful evolution and ever ready to help in further advances? Why don't they make an onslaught on national and individual health improvement where its roots lie? Here is a five-point national health programme which will cost the Australian Government one-tenth of the cost of a nationalized medical service, which will by raising nutrition standards and housing standards prevent disease, and which, so far from destroying the present efficient organization, will build up on it and around it:

1. Give the people enough of the right food and teach them through radio and Press what it is.
2. Give every family a decent house to live in.
3. Enforce by law immunization where it is known to be effective.
4. Organize and subsidize through competent bodies popular medical education.
5. Organize and subsidize throughout Australia X-ray, pathological and biochemical departments so that every practising doctor even in the most remote parts could raise his standard of diagnosis, which is after all the basis of medical efficiency.

On the whole question, then, there can be no surrender nor any betrayal of an efficiency built up patiently over the years. There can be no Vichy. We are descendants of centuries of ancestors who have struggled and survived by the Hippocratic conscience. That it is which fortifies us in our daily work. May it never so happen that that will be replaced by the cold power of a machine!

Yours, etc.,

H. R. R. GRIEVE.

113, Homer Street,
Undercliffe,
New South Wales.

November 6, 1941.

## A NATIONAL MEDICAL SERVICE.

SIR: Dr. Dark complains that I put forward opinions rather than facts on the question of socialization of medicine. The realities of such a system only become facts once the machinery tries to work, and at present one is limited to trying to visualize what would happen. The essence of the problem, as I see it, concerns general practice chiefly and is whether the standard of service to the people is better under our present system or under the proposed State bureaucracy. No one denies that there are imperfections in the present system, but are we going to scrap it for a new system which perpetuates most of the imperfections of the old, promises to destroy most of its virtues and substitutes a very doubtful and hazardous experiment in socialism?

To visualize the difference between the respective services offered by private enterprise on one hand and State undertakings on the other, let anyone first approach the counter of a progressive competitive departmental store and then approach the counter of any non-competitive Government-controlled office, like the post office for example. The heart of a public servant is seldom stirred to compassion by any considerations of urgency or hardship in your affairs. He doesn't get any medals for violently disturbing the routine of the machine, and I see no medals being handed out under State medicine to the conscientious doctor who does a particularly good job.

To get a foretaste of medical socialism in action one has only to become a cog in the government-run machine called the Army, and to watch the high degree of medical "service" dispensed on sick parades in our Militia camps. As in the proposed State-run service here every man has the right to demand medical attention for any conceivable ailment, small or large, real or imaginary, honest or tendentious, and—this is the essential point—at no immediate cost to himself. Because of the absence of the sobering effect of a fee the most difficult and time-consuming part of the interview is wasted in assessing just how sick or sore he really feels. The reaction in the doctor is axiomatic—and the ideals of true service to one's "suffering" fellow man are inexorably worn done in the stoutest altruist. Omitting outback patients (who would fare no better under socialization), would some malcontent let us know just what patients are being denied the best medical aid under the present system? The poorest hobo can, and very often does, receive the most skilful treatment the most eminent men of our profession can give and without the cost of a penny. Those members of the community who prefer a form of pseudo-socialism in medical service, already have the opportunity of indulging their preferences in a friendly society contract practice. Those who can afford to pay are asked to pay for maintenance and repair of their most cherished possession, very often much less than they cheerfuly lavish on their radio sets or motor cars.

Dr. Dark exemplifies Pavlov and Filov as products of socialism—which I question on the grounds of historical and material fact—but he has brought forward no reason whatever why the present "old order" cannot continue to produce its Pasteurs, Jenners, Listers, Kochs, Bantings, Ehrlichs, Rosses and Mackenzies *ad infinitum* as it has in the past. The greatest difficulties these men had to fight were not scientific problems, but standardized, stereotyped, "accepted" schools of thought. Under State-run medicine I can imagine certain schools of thought receiving the official imprimatur of governmental orthodoxy and us, as State servants, obliged to conform as so many servile sheep, or

run the gamut of intolerable inquisitions. Dr. Dark states that he takes it that "the incentives to good work are not material reward, but the mere satisfaction of knowing that work is being well done and the approval of one's fellows for that reason". I do not doubt Dr. Dark's sincerity nor his altruism, and I honestly admire those who hold and practise these ideals—but the thoroughness of their realization varies with the degree of human frailty in our individual make-ups. I feel that the question of material reward will only cease to come into one's work when we rise above the instincts of normal human beings and become saints. A halo will then become the substitute for material reward. Frankly, I have no ambition to wear a halo. I just simply prefer (and I think with all fairness) a fair and reasonable remuneration for the most highly skilled, difficult, arduous, responsible and wearying task to which a man can set his hand, and I have no compunction in accepting it.

The present system of private practice and general service to the public has grown *pari passu* with the development of democracy itself and, like democracy, is founded on an unwritten charter of rights and responsibilities between patient and doctor and, like democracy, no doubt it has its imperfections. It has been a spontaneous evolutionary process, and I still have to see advanced one good reason why we should suddenly decide to destroy its structure and replace it by a revolutionary "new order" of medical socialism.

To those who, like Dr. Maude, object to my "robustness", kindly allow me to quote from the published statements of the leaders of our profession in New Zealand on their scheme:

Sir James Elliott: "All men who love freedom and justice must take the buttons off their foils and fight for the right whatever may be the consequences." "Two classes will operate the scheme, the Brahmin and the coolie class. The Brahmin will be the ruling class. They are the Minister of Health, his officers and his clerks. These will enjoy good salaries, a forty-hour week, all public and annual holidays, the right of advancement, pay on sick leave and at last a pension. But the coolie class, that is the practising profession, will get none of these. While the Brahmin class lie warm in bed dreaming of their comfortable security and certain pension, the coolie doctor may be called out at 2 a.m., utterly exhausted after a heavy day, to give highly technical advice and treatment for which he will receive in due course, and all the necessary forms completed, not enough to pay for the depreciation on his car for the journey. A taxi-driver would get twice as much and more for his part of the job."

"Why should a learned and public-spirited class of the people be singled out and forced to give up their constitutional rights as free citizens or else be starved or imprisoned?"

Dr. H. M. Wilson, President of the New Zealand Branch of the British Medical Association: "It substitutes conscription for private rights as a permanent feature of medical practice in peace as well as war. It is a system which no government would wish or dare to impose upon the weakest or least significant trade unions in the Dominion."

It is the duty of those who are best able to visualize the retrograde evils of the "new order" to follow the shining example of our New Zealand brothers and resist to the utmost the schemes of politicians who want to feed political sop to a gullible and undiscerning public. No less is it the duty of us at home to see that those of our members who are fighting abroad for the democratic way of life do not come home to find that a system of medical fascism has been foisted on them in their absence.

Yours, etc.,

R. D. DAVEY.

Croydon Park,
New South Wales,
November 8, 1941.

# Naval, Military and Air Force.

## APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Number 215, of October 30, 1941.

### AUSTRALIAN IMPERIAL FORCE.

*Australian Army Medical Corps.*

Major E. E. Dunlop is appointed to command, temporarily, a Casualty Clearing Station, *vice* Major W. Park, 18th July, 1941.

*To be Major.*—Captain J. Ray, 19th August, 1941.

*Permanent Supernumerary List.*

Captain J. J. Ryan, M.C., is transferred from the Australian Army Medical Corps, 18th July, 1941.

### AUSTRALIAN MILITARY FORCES.

AUSTRALIAN ARMY MEDICAL CORPS.

*Seventh Military District.*

*To be Major (temporarily).*—Captain W. K. W. Flook, 16th April, 1941.

**Northern Command.**

*First Military District.*

The following officers are appointed from the Reserve of Officers (A.A.M.C.) and to be Captains (provisionally): Honorary Captains C. W. Taylor, 28th February, 1941, and S. Gohstand, 23rd September, 1941.

*To be Honorary Captains.*—Arthur John McLaren Ross, 22nd September, 1941; and Peter Bede English, 28th September, 1941.

**Eastern Command.**

*Second Military District.*

The following officers are appointed from the Reserve of Officers (A.A.M.C.): Honorary Captain J. M. Yeates and to be Captain (provisionally) and Captain J. F. Lipscomb, 9th July, 1940.

**Southern Command.**

*Third Military District.*

Captain A. Weigall is appointed from the Reserve of Officers (A.A.M.C.), 14th April, 1941 (in lieu of the notification respecting this officer which appeared in Executive Minute No. 111/1941, promulgated in *Commonwealth Gazette* No. 124 of 1941).

### ROYAL AUSTRALIAN AIR FORCE.

*Permanent Air Force: Medical Branch.*

Temporary Squadron Leader E. H. Anderson is granted the acting rank of Wing Commander whilst employed as a Wing Commander, with effect from 1st October, 1941.

Flight Lieutenant J. M. Casley is promoted to Temporary Squadron Leader, with effect from 1st October, 1941.

Temporary Squadron Leader W. L. B. Stephens is granted the acting rank of Wing Commander whilst employed as a Wing Commander, with effect from 13th October, 1941.

*Citizen Air Force: Medical Branch.*

Temporary Squadron Leader J. G. Brown is granted the acting rank of Wing Commander whilst employed as a Wing Commander, with effect from 1st October, 1941.

The following Flight Lieutenants (Acting Squadron Leaders) are promoted to temporary Squadron Leaders, with effect from 1st October, 1941: J. L. Walter, R. G. Morris, D. E. Davies, A. N. Poulton.—(Ex. Min. No. 148—Approved 29th October, 1941.)

Temporary Flight Lieutenant A. W. Raymond is granted the acting rank of Squadron Leader whilst employed as a Squadron Leader, with effect from 1st October, 1941.—(Ex. Min. No. 149—Approved 29th October, 1941.)

The following are appointed to commissions on probation with the rank of Flight Lieutenant, with effect from the dates indicated: Rory Clevedon Willis, M.B., B.S., 2nd September, 1941; Vernon James Odlum, M.B., B.S., 15th September, 1941; John Stephen Boxall, M.B., B.S., 18th September, 1941.

*Reserve: Medical Branch.*

Basil Nicholas Orr Colahan, M.B., B.S., is appointed to a commission on probation with the rank of Flight Lieutenant, with effect from 19th September, 1941.

The probationary appointment of Flight Lieutenant F. T. Hartnett is terminated, with effect from 19th August, 1941.—(Ex. Min. No. 151—Approved, 29th October, 1941.)

## CASUALTIES.

ACCORDING to the casualty list received on November 6, 1941, Captain N. R. Godby, A.A.M.C., of East St. Kilda, Victoria, previously reported missing, is now reported prisoner of war.

# Obituary.

## JAMES LEE SELWOOD.

DR. JAMES LEE SELWOOD, whose death was recently announced in these pages, was born on June 18, 1887, at Queenstown, Victoria. His father was James Thomas Selwood, inspector of police, Melbourne.

James Selwood distinguished himself from the beginning of his school days by always being first in class and he was always considered a most solid student. He was outstanding at lacrosse and tennis. He entered the medical school at Melbourne in 1906, taking first-class honours in the examination for physics and chemistry. He graduated Bachelor of Medicine and Bachelor of Surgery in 1911. He held resident positions at the Melbourne Hospital, the Brisbane General Hospital, the Brisbane Children's Hospital, the Brisbane Women's (Lady Bowen) Hospital. He practised at Lisburn Street, East Brisbane, as a general practitioner. He was honorary surgeon to the Brisbane Children's Hospital for five years, from 1917 to 1922, honorary physician to the Lady Bowen Hospital for seven years, from 1917 to 1924, and honorary surgeon to the Mater Misericordiæ Public Hospital for one year, 1921. James Selwood acted as camp and general medical officer at Maryborough during the 1914-1918 war, and was senior medical officer at Redbank from the declaration of war in 1939 until he died. Before entering camp he had a large private and lodge practice, which was conducted at Five Ways, East Brisbane. He was an enthusiastic member of the British Medical Association and of the Queensland Branch council. His hobby was bowls, and he was a foundation member of the East Brisbane Bowling Club. He was also lacrosse blue in his younger days in Melbourne. He was a member of the United Service Club, Brisbane Club, the Constitutional Club and the Johnsonian Club.

Dr. Selwood was a great reader and found his relaxation in his library. He leaves a wife and two daughters to mourn his tragic and sudden passing.

Dr. L. P. Winterbotham writes:

For many years I had the pleasure of a close association with my friend Major Selwood. We played together at the university and started practice in South Brisbane almost at the same time. He was a fine man, always able to enjoy and tell a good joke, and with a keen sense of humour that many a time stood him in good stead. For several years he was a member of the Branch council, and for a good many more he was one of the solid members of the Contract Practice Committee; in both positions his counsel was well considered, moderate and much thought of. His last two years were spent in camp at Redbank, and his continual grouch was that he was not picked to go overseas. I knew his friends thought highly of him and the children loved him, and I think it will take many of his old friends in Brisbane a long time to get over the shock of his sudden and unexpected death.

### ROBERT MENZIES KINROSS.

We regret to announce the death of Dr. Robert Menzies Kinross, which occurred on November 5, 1941, at Artarmon, New South Wales.

### WILFRED KENT HUGHES.

We regret to announce the death of Dr. Wilfred Kent Hughes, which occurred on November 8, 1941, at Warrandyte, Victoria.

## Nominations and Elections.

The undermentioned has applied for election as a member of the New South Wales Branch of the British Medical Association:

Peterson, Bruce Henry, M.B., B.S., 1941 (Univ. Sydney), Sydney Hospital, Sydney.

## Corrigendum.

In the leading article in the issue of October 25, 1941, on microradiography and the tuberculosis problem in Australia, reference was made to the health centre in Prahran, Victoria. It was stated that this health centre is part of a "complete central organization for 'total' health" conceived by Dr. R. H. Fetherston, Health Officer of the City of Prahran, and described in the *Health Bulletin* of the Victorian Department for Public Health for January-June, 1941. The wording of the sentence has created the impression that the words quoted in inverted commas are Dr. Fetherston's words. Dr. Fetherston writes that he has never conceived any scheme for total health. The words are taken from an unsigned article in the *Health Bulletin*. We offer our apologies to Dr. Fetherston for any ambiguity in the sentence in question.

### MEDICAL WAR RELIEF FUND.

The following is a twentieth list of contributions to the Medical War Relief Fund established by the Federal Council of the British Medical Association in Australia for the relief of distressed medical practitioners in Great Britain.

**Victoria.**

£10 10s.: Dr. T. O. Sayle.

£5 5s.: Dr. R. E. Allen, Dr. Francis E. McAree, Dr. Sidney F. Ridley, Dr. G. E. M. Scott.

£3 3s.: Dr. M. H. Box.

£2 2s.: Dr. F. G. Donovan, Dr. W. Godbehear, Dr. E. C. Varley.

## Diary for the Month.

Nov. 19.—Western Australian Branch, B.M.A.: Branch.
Nov. 26.—Victorian Branch, B.M.A.: Council.
Nov. 27.—New South Wales Branch, B.M.A.: Branch.
Nov. 27.—South Australian Branch, B.M.A.: Branch.
Nov. 28.—Queensland Branch, B.M.A.: Council.
Nov. 28.—Tasmanian Branch, B.M.A.: Council.
Dec. 3.—Western Australian Branch, B.M.A.: Council.
Dec. 3.—Victorian Branch, B.M.A.: Branch.
Dec. 3.—Victorian Branch, B.M.A.: Council.
Dec. 4.—South Australian Branch, B.M.A.: Council.
Dec. 9.—Tasmanian Branch, B.M.A.: Branch.

## Medical Appointments: Important Notice.

Medical practitioners are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

*New South Wales Branch* (Honorary Secretary, 135, Macquarie Street, Sydney): Australian Natives' Association; Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phœnix Mutual Provident Society.

*Victorian Branch* (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federated Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

*Queensland Branch* (Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

*South Australian Branch* (Honorary Secretary, 178, North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

*Western Australian Branch* (Honorary Secretary, 205, Saint George's Terrace, Perth): Wiluna Hospital; all Contract Practice appointments in Western Australia.

## Editorial Notices.

Manuscripts forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to The Medical Journal of Australia alone, unless the contrary be stated.

All communications should be addressed to the Editor, The Medical Journal of Australia, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2.)

Members and subscribers are requested to notify the Manager, The Medical Journal of Australia, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such a notification is received within one month.

Subscription Rates.—Medical students and others not receiving The Medical Journal of Australia in virtue of membership of the Branches of the British Medical Association in the Commonwealth can become subscribers to the journal by applying to the Manager or through the usual agents and booksellers. Subscriptions can commence at the beginning of any quarter and are renewable on December 31. The rates are £1 for Australia and £2 5s. abroad *per annum* payable in advance.